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VOLUME XV NOVEMBER 1958 NUMBER 6

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VOLUME XV

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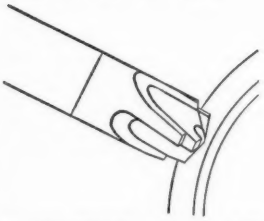

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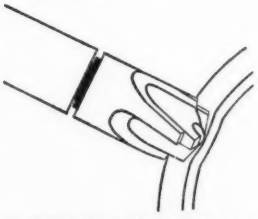

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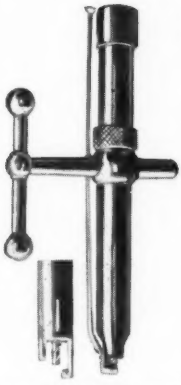

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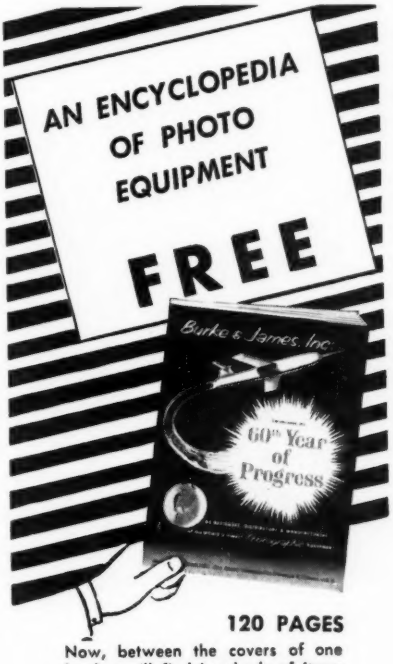

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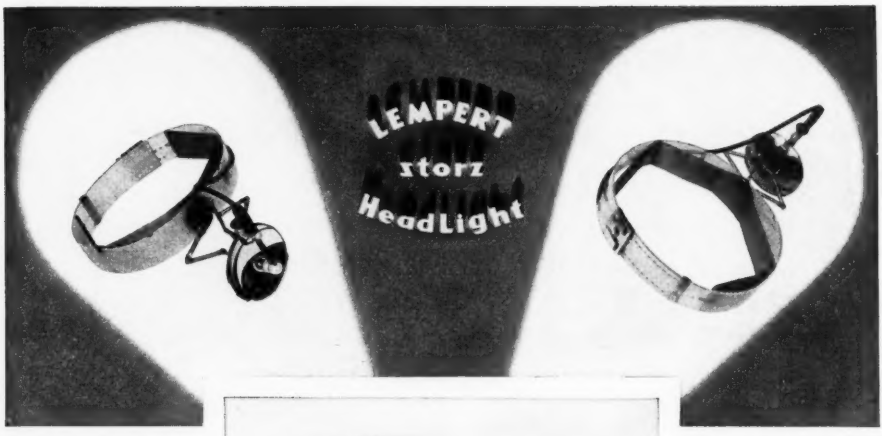

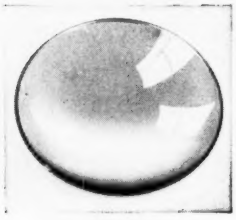

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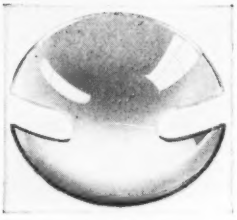

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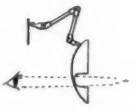

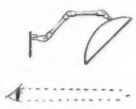

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Atlas of TUMORS of the NERVOUS SYSTEM

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Attending Neurological Surgeon, Montefiore Hospital, and Professor and Chairman, Department of Surgery, Albert Einstein College of Medicine, New York City

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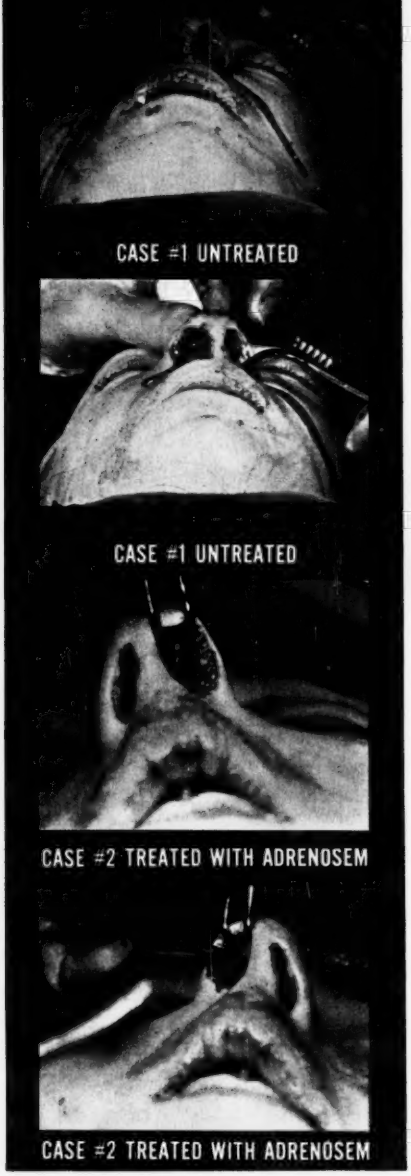

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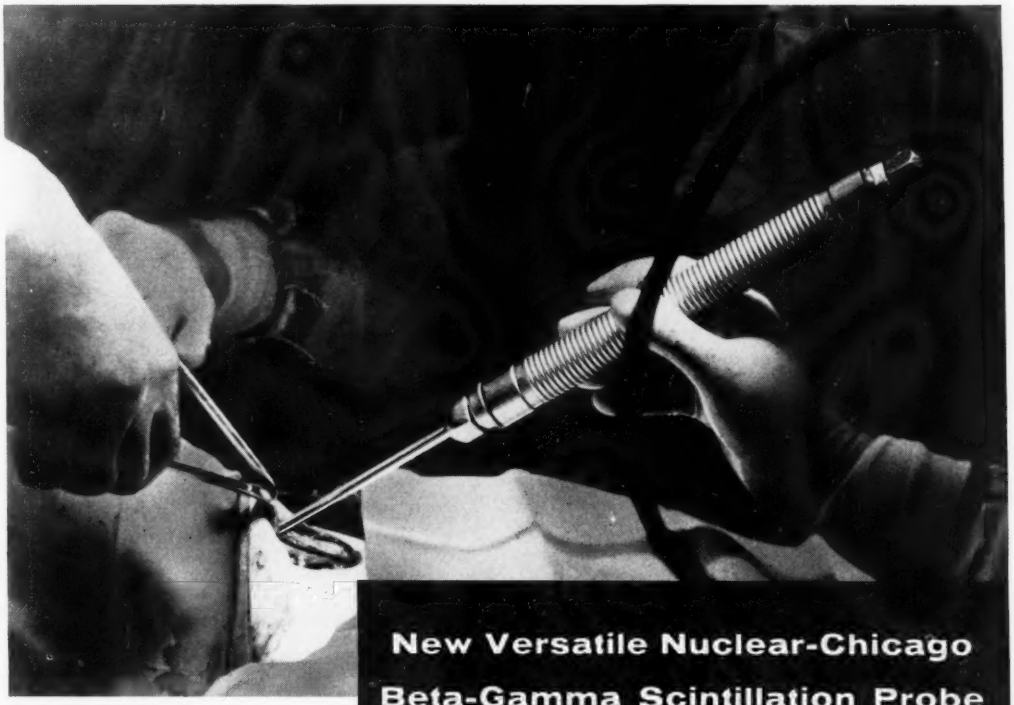

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Preface

The relationship of brain and consciousness can be considered from the epistemological as well as from the neurological aspect. In this disquisition, I shall attempt to discuss both points of view, and to integrate these aspects into a unified presentation.

The purely neurologic subject matter constitutes the greater part, approximately two thirds of this presentation, and its validity or purpose retains, for practical intents, an intrinsic independence of any epistemologic interpretation.

Nevertheless, I believe that such an epistemologic interpretation, frequently avoided by the neurologist, is of substantial theoretical importance. Many authors evade the question by slurring over the crux at issue with a few generalities concealing the gist of the problem. However, some writers, conversant with the topic, have clearly recognized the elementary and essential fact that the experienced private perceptual space is not identical with the inferred public physical space. This recognition, of course, should imply the distinction of the two different space-time systems including their dimensions.

It has also been stated—not without justification—that the relation of brain and consciousness is to some extent a semantic problem. In this respect, the inherent deceptiveness of abstractions and of apparent verbal precision must be kept in mind: *fraus latet in generalibus.* While in theoretical systems of logic based upon digital rules, the principle of excluded middle can be considered valid, this principle is certainly not valid in many aspects of applied scientific and practical everyday thinking which operates rather according to analogue rules: Truth value is here not digital or binary, corresponding exclusively to one of the two values zero or one, but encompasses, within these two limiting values, innumerable intermediates between truth, that is significant conformity, and error, fallacy or falsity. *H. K.*

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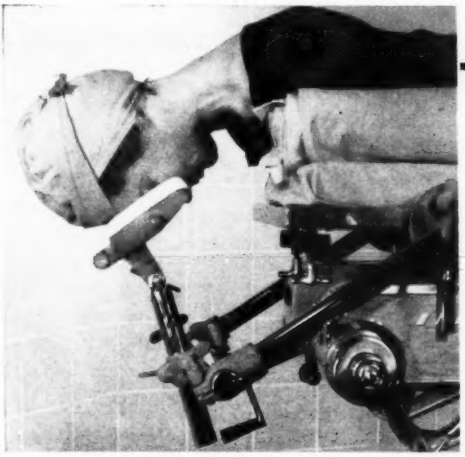

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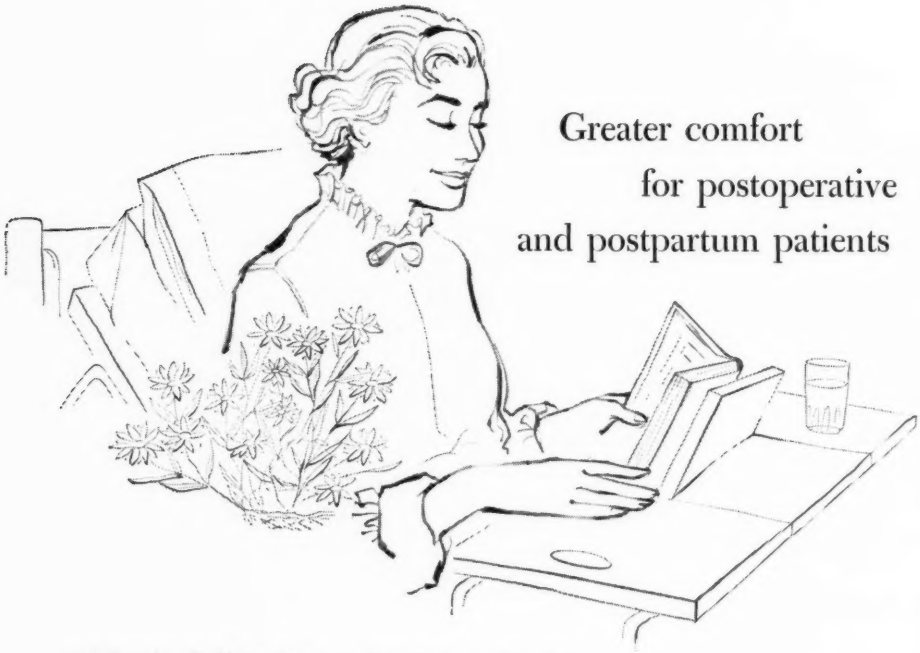

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THE HARVEY CUSHING SOCIETY

PAST, PRESENT AND FUTURE*

HOWARD A. BROWN, M.D.†
University of California, San Francisco, California

(Received for publication May 19, 1958)

It is grateful and sincere appreciation that I extend to the members of the Harvey Cushing Society for the honor that has been extended me in allowing me to represent you as your president during the past year. It has been a very pleasant task, and it has been made doubly so by the excellent cooperation of the other officers and of the committees of the society who have given so much time and effort to make this an outstanding meeting. May I assure you that election to this office constitutes a very great honor and marks, for me, the highest point in my career in the field of neurological surgery.

We are now embarking upon the second quarter century since the founding of the Harvey Cushing Society, and it would therefore seem appropriate at this time to review the founding and early days of this organization; to trace its development, growth and accomplishments during the past twenty-five years, to evaluate its present status; and surely most important of all, look ahead to our future—its goals and the policies we must pursue in achieving them.

First, let us examine the reasons for the formation of this particular group. The Society of Neurological Surgeons was founded by eleven neurosurgeons in 1920 and was, prior to the founding of the Harvey Cushing Society, the only one in the United States representing this particular specialty. The membership maintained in this group was restricted to a small number despite the rapid growth of neurological surgery during the next ten years. Many of the younger neurosurgeons keenly felt the need for contact with their colleagues in order to effect an exchange of ideas that would advance their knowledge and improve their skills in this chosen field. On June 24, 1931, Doctor R. Glen Spurling wrote to Doctor W. P. Van Wagenen suggesting the formation of a new society and mentioning the names of other neurosurgeons who might be interested. Following further correspondence, a meeting was held at the Hotel Raleigh in Washington, D.C., on October 10, 1931 and was attended by Doctors Temple Fay, Eustace Semmes, R. Glen Spurling, and W. P. Van Wagenen. The decision was made to form a new society, the preliminary purposes and plans, including establishment of a central brain tumor registry, were discussed and names of poten-

* Presidential address, read at the Twenty-sixth Anniversary Meeting of the Harvey Cushing Society, Washington, D.C., April 22, 1958.

† 384 Post Street, San Francisco 8, California.

tial members were considered. Doctor Harvey Cushing was advised of the proposed formation of the new group, and he expressed his approval and invited the group to hold their first meeting at his clinic.

Thirty charter members were chosen and the first meeting was held in Boston, May 6, 1932, with twenty-three members of the charter group present. The society was named in honor of its patron, Doctor Harvey Cushing. Doctor William P. Van Wagenen was elected the first president of the Harvey Cushing Society.

At the beginning, the purpose of this society was the promotion and the advancement of the various fields of organic neurology, and this was changed later to the advancement of neurological surgery. The active membership at the beginning of the society included not only neurosurgeons but those in the fields of neurology, neuroanatomy, neuro-ophthalmology, neuropathology, psychiatry, psychology, roentgenology, and other scientific works bearing on neurology. Subsequently, the neurosurgeons have constituted the active membership and the members of the allied fields have become associate members of the society.

FIG. 1. Dr. W. P. Van Wagenen, Dr. R. Glen Spurling and Dr. R. Eustace Semmes—three of the four original founders of the society.

In order to have closer contact and allow an exchange of ideas it was felt that a small group was desirable, and the society was numerically limited to thirty-five members at the outset. The first program included various scientific papers, and the presentation of operative cases by Doctor Cushing. I could not be otherwise than amused by the title of the paper given by Doctor Stafford Warren who is now the Dean of the University of California School of Medicine at Los Angeles. The name of this paper was the "Treatment of Diseases of the Nervous System by Hyperthermia." In the past twenty-five years the mercury has fallen. The emphasis is now at the other end of the thermometer.

A diary note of one of the members at the first meeting gives us a few interesting details and sidelights of the meeting. "The membership contains men from all parts of the country. There are two from Chicago, one from Kansas City, one from the Pacific Coast, and a scattering from the Eastern seaboard. Doctor Cushing received them this morning at ten o'clock, and he said that he felt like an obstetrician bringing a new and protesting offspring into existence. He welcomed them warmly, nevertheless, asking them only to remember that in ten years' time another group would be coming along that would look upon the present one as senile and antiquated."

We see here Doctor W. P. Van Wagenen, Doctor Glen Spurling and Doctor Eustace Semmes, three of the four original founders of the society (Fig.

FIG. 2. Dr. Cushing conducting an operative clinic at the first meeting of the society.

1). I was not successful in coaxing a photograph from the fourth member, Doctor Temple Fay.

Doctor Cushing conducted an operative clinic for the society at the first meeting in Boston (Fig. 2). Here he is wearing the traditional headlight used in neurosurgical operations. Dick Light's comment about the headlight was amusing, "It was a very poor source of illumination, but an excellent radiant and heat element. Anybody touching it got burned."

This picture is of the charter members attending the first meeting (Fig. 3). Doctor Cushing wrote to Doctor Tracy Putnam, the secretary of the society, on June 15, 1932: "Dear Tracy, Thanks much for your note and for

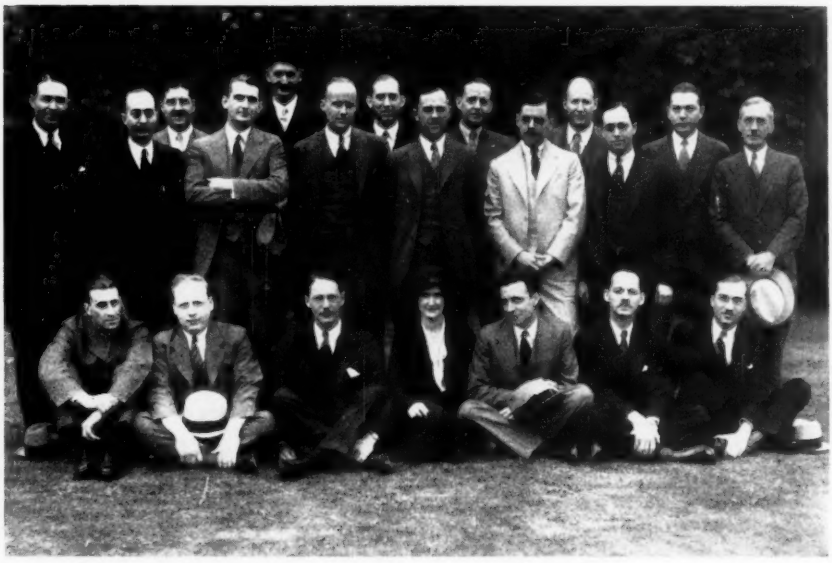

FIG. 3. Charter members attending the first meeting in Boston in 1932.

the photograph of the newborn club. They look like a very serious and hard-working group, all except the three squatting figures in the center of the picture. Now, if you had been sitting where John is, you could have perhaps cooked up as coy a smile as he was able to manage. I am very proud of all of you, and the fact that I should be immortalized by having you use my name is a source of pride and gratification. Always affectionately yours, Harvey Cushing."

There is an interesting bit of history in connection with the name of the society that is not generally known. It was Doctor Temple Fay who suggested the name "Harvey Cushing" for the society. After the society had been in existence for about ten years, a move was made to change the name inasmuch as the society was composed of many more than the graduates of

FIG. 4. Dr. Cushing at his desk in the Brigham Hospital.

FIG. 5. Dr. Cushing sketching the operative findings while having tea in his dressing room.

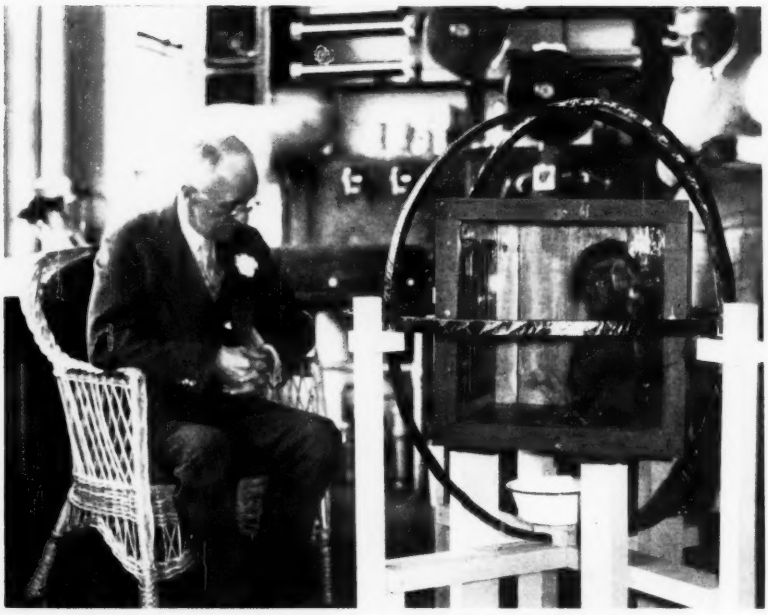

FIG. 6. Dr. Cushing watching a monkey during a convulsive seizure in the laboratory.

the Cushing Clinic and inasmuch as Doctor Cushing was not personally responsible for the formation of the society. The idea seemed to be that a "name" was objectionable. However, it was pointed out that several outstanding institutions had survived being named after such characters as John Harvard, Eli Yale, William Penn, Ezra Cornell, Jesus Christ, Mohammed, Brigham Young, and so on. And the "name" remained unchanged.

The following pictures (Figs. 4–6) show Doctor Cushing in various phases of his work.

"Tea time was almost a ritual with Doctor Cushing, and the tea hour with Doctor and Mrs. Cushing in their home was for their guests a memory in hospitality. Although Doctor Cushing ate sparingly, he insisted upon his toast and tea at four in the afternoon and was the perfect host whether at a croquet match or in his dressing room at the hospital."

The second meeting was held in Louisville, and because of the efforts of the society the Brain Tumor Registry was set up under the able direction of Doctor Louise Eisenhardt who has cared for this most important project during the past twenty-five years.

Here we have a view of the Surgical Laboratory at the Peter Bent Brigham Hospital in Boston and Doctor Eisenhardt (Fig. 7).

The following is a view of the eighteen members who attended the Louisville meeting in 1933 (Fig. 8). The next view includes some very pleasant

FIG. 7. Dr. Louise Eisenhardt at work in the Surgical Laboratory.

scenery. It seems that considerable progress has been made, at least along certain lines, and instead of tea we now have a picture of the members partaking of something a mite more fortifying and, while I will withhold a prognosis, I can say that perhaps this is a hint of things to come in the Harvey Cushing Society. Here we have some members and some mint juleps at Louisville (Fig. 9).

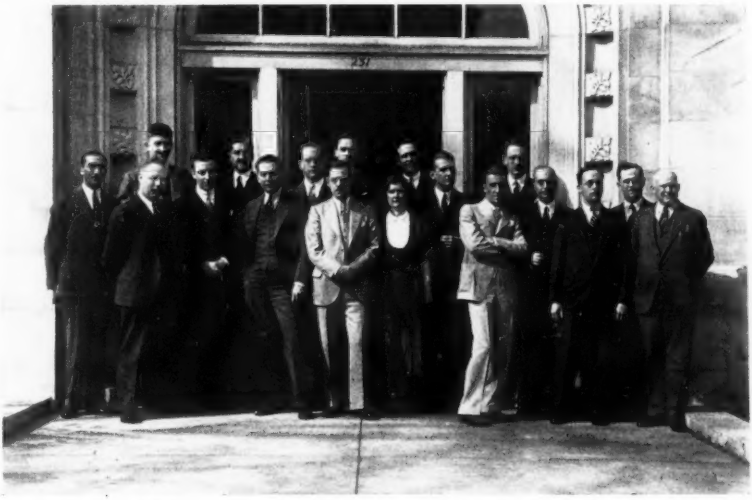

FIG. 8. The second meeting of the society, Louisville, Kentucky, 1933.

FIG. 9. Some members and some mint juleps at Louisville.

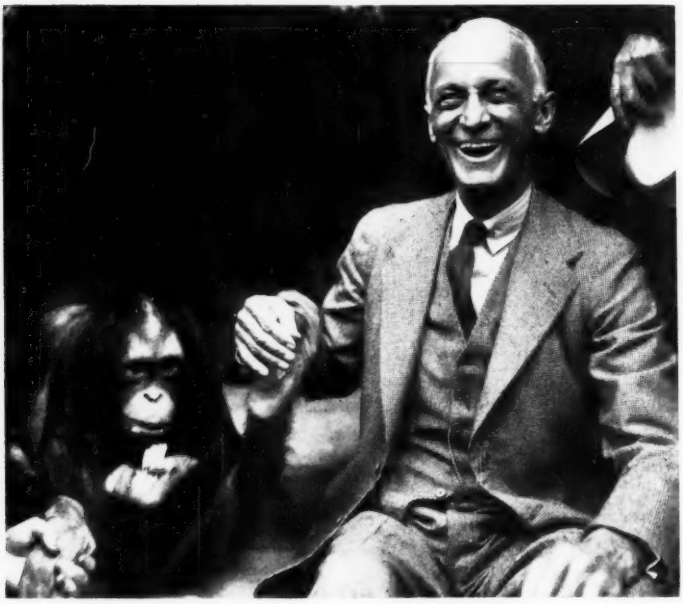

FIG. 10. Dr. Cushing and Chimpanzee Lulu, New Haven, 1935.

FIG. 11. Dr. Ernest Sachs.

The next two meetings were held in St. Louis and in New Haven with an increased membership level to fifty and a required three-fourths affirmative vote to elect.

I refuse to give any commentary at all on the next picture, taken at the 1935 meeting in New Haven (Fig. 10), but perhaps a little information about the two distinguished citizens shown here would be in order. Doctor Cushing you all recognize, and the other is Lulu.

In 1936 we met at Rochester, and Doctor Ernest Sachs (Fig. 11) was elected to honorary membership, the first to follow Doctor Cushing.

Doctor Sachs told the society that Doctor Cushing had taught him that, "The way to keep young is to keep in contact with young men." He also expressed the wish that he " . . . might live long enough to see in twenty-five years what extraordinary things you and your students will be doing in neurosurgery." We are delighted to note that he has already watched twenty-two of those years with their fruitful developments.

In Philadelphia at the 1937 meeting there was much discussion about the size of the society and reports were at hand that a third neurosurgical group was in the process of being formed. The suggestion was made that active members be retired after seven years but that they be allowed to attend meetings and to dine as senior members. John Fulton was then the President and went on record with some very pertinent comments concerning the

proposed farm system for the more mature element of the membership. He wrote as follows: "Senior members can, of course, dine, and that is probably all they would be good for. It would be better to chloroform the senior members than to rally them. In short, I am not very much in sympathy with the idea of retiring people after seven years and then trying to keep them in good humor with dinners and rallies, and invitations to speak now and again at meetings. Such an arrangement would, it seems to me, ruin the present spirit of the society." An outstanding event of this meeting, and I have no way of knowing if it were in any way connected with the controversial issue of how to dispose of the older members, was John Fulton's successful efforts to intoxicate some pet trout in Doctor Temple Fay's pool with martini cocktails. Louise Eisenhardt has recorded the official resolution relative to the event and the spontaneous death of six drunken trout on the edge of the pool, discovered early the next morning. There is no mention in the official record of the condition of the neurosurgeons on the following morning.

At Memphis in 1938 there was much discussion concerning the establishment of a neurosurgical board, and a report favoring the formation of such a board was given by Doctor Winchell McK. Craig. Abandoned at this meeting was the proposed formation of regional neurosurgical societies under the sponsorship of the Harvey Cushing Society.

Our eighth meeting at New Haven marked an historical date in the progress of the society. At this time, on April 18, 1939, we celebrated the seventieth birthday of Doctor Cushing. On this happy occasion, Doctor Louise Eisenhardt, president of the society, presided as toastmistress when a host of Doctor Cushing's friends, pupils and admirers paid tribute in person, by letter and telegram to the "Chief."

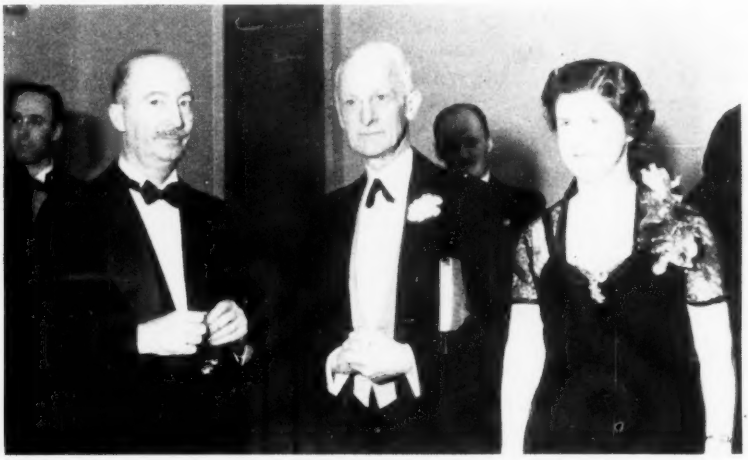

FIG. 12. Dr. Semmes, President-Elect, Dr. Cushing, and Dr. Eisenhardt, President, New Haven, 1939.

FIG. 13. Dr. Cushing expresses his pleasure over the birthday cake, while Dr. Arnold Klebs smilingly looks on. New Haven, 1939.

On behalf of the society, Doctor Eisenhardt presented him with a complete bibliography of his own writings. A few of the pictures taken at that time remind us of his last official meeting with the Harvey Cushing Society. The first view is of Doctor Cushing, Doctor Eisenhardt, president, and Doctor Eustace Semmes, president-elect (Fig. 12). The next view is of Doctor Cushing's birthday cake (Fig. 13).

It is with regret that I mention the next important date in the history of the society, October 7, 1939, which marks the death of Doctor Harvey Cushing (Fig. 14).

He had pioneered our specialty, and he had inspired and taught an entire generation of doctors. His friends and admirers were countless, and his contribution to the medical profession is legend. His fraternity was mankind. I would like to read a paragraph from a letter written by Doctor Paul Bucy to Doctor Louise Eisenhardt following Doctor Cushing's death. "Death comes to *all* men. But few of us can look back upon a career of worthwhile endeavors. And fewer still are permitted the opportunity to see the world-wide ripening of the fruits of their labors. Yet to Harvey Cushing all these were accorded. We cannot begrudge him his rest."

The American Board of Neurological Surgery was incorporated on August 1, 1940 and the following three members of the Harvey Cushing Society

FIG. 14. Dr. Harvey Cushing.

were appointed to it: Doctors Leo Davidoff, Temple Fay and Winchell McK. Craig. This society has continued to participate in the important functions of this board since that time.

At the meeting in Kansas City in 1940, there was further discussion of expansion, and a committee was appointed to report on this important problem in 1941. A somewhat solemn note prevailed at this meeting because of the recent death of Doctor Cushing, and the society paid its respects to his memory in a very appropriate ceremony at the formal banquet. There were now fifty-two active members, and here we see a picture of that group (Fig. 15).

The report of the committee rendered at the Rochester, New York meeting in 1941 recommended that the society be expanded into a neurosurgical organization of national scope, that the membership include repre-

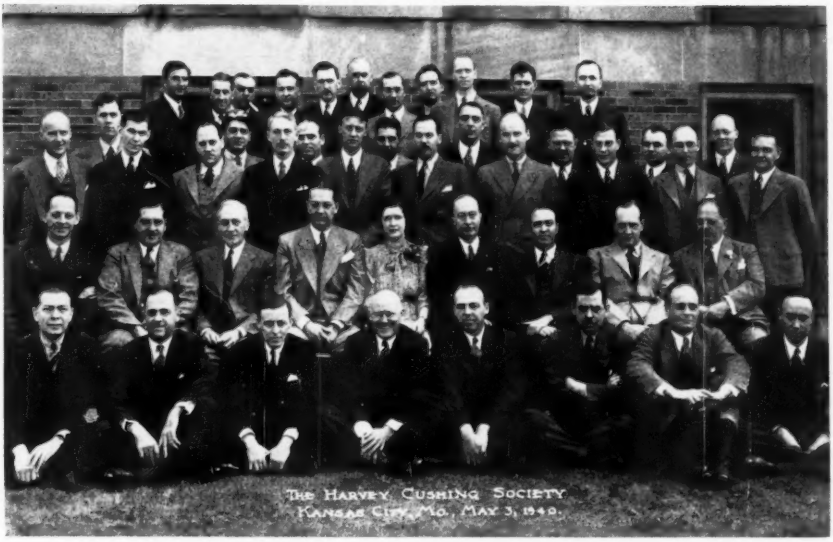

FIG. 15. Kansas City, Missouri meeting, 1940.

sentative members from related fields, and that the members of the other two neurosurgical societies be admitted to membership. All active members of the Harvey Cushing Society must be certified by the American Board of Neurological Surgery.

The necessary changes in the by-laws were made at New York City in 1942, and the real expansion of the society began. Who could have dreamed of the veritable legion of neurosurgeons to come? I am reminded here of a remark made by one of the charter members after his return from a recent meeting of the Harvey Cushing Society at which time he said he was continually amazed at the large number of members and commented that he wondered if any one had found out yet what was causing neurosurgeons.

At New York in 1943 it was recommended that a Journal of Neurosurgery be established under the auspices of the Harvey Cushing Society. An editorial board was appointed under the chairmanship of the late Doctor Gilbert Horrax. The Journal started publication on a bi-monthly basis in 1944 and has been a success from the beginning. It has constantly been improved under the guiding hands of Doctor Louise Eisenhardt and the editorial board.

The war years caused curtailment and finally cancellation of the 1945 meeting. Many of our members served in the armed forces at home and abroad, bringing great credit to the field of neurosurgery.

A post-war reunion was held in Boston in 1946. At this time the members of the Society of Neurological Surgeons were elected to membership *en bloc*.

In 1947 the society met in Hot Springs, Virginia. Here is a photograph of the past presidents at Hot Springs (Fig. 16).

By 1949, there were one hundred eighty-eight members, and changes were made in the by-laws in 1951 providing for a president-elect to give greater efficiency in the activities of the society. A membership committee was formed to carry out the major task involved in screening the many applications for membership each year.

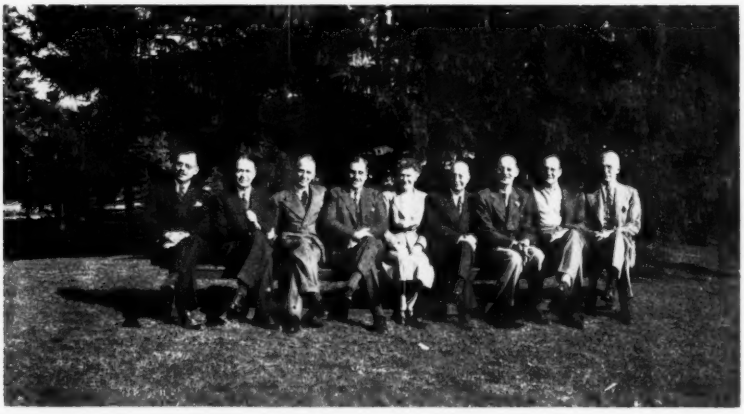

FIG. 16. Past presidents, Hot Springs, Virginia, 1947. Drs. Van Wagenen, Spurling, McKenzie, Fay, Eisenhardt, Semmes, Putnam, Fincher and Ingraham.

There is no doubt about the role our *ladies* have played in helping to build and to strengthen our society. With their cooperation, we have been able to hold our meetings at many different and sometimes quite distant places which has indeed added to the over-all interest in the attendance. A sort of "Join the Harvey Cushing Society and See the World" or "Have Wife—Will Travel" operation. The ladies were officially recognized by our group when the Ladies Auxiliary was added to the Constitution in 1951. Seriously, the ladies have contributed much help since they were first organized with Mrs. Paul Bucy acting as their first president. We are very appreciative of their support and of their infinite patience with their neurosurgeons. Most of all, we are deeply appreciative of their charm. We welcome them here today and wish them many happy years to come with the organization.

Neurosurgical training since the end of World War II has expanded at an amazing rate, and the number of trained neurosurgeons in this country has grown at approximately sixty to seventy a year as indicated by the figures of the American Board of Neurological Surgery. The membership in this society has increased steadily and by 1952 there were three hundred forty-two members listed. Even the long trek to Honolulu enticed a goodly number to the enchanting islands of the Pacific—a meeting that will long be remembered by those present.

Now we have grown to an over-all membership of 593. It would seem

that we have increased as efficiently as bacteria in five-day-old potato salad, and it is certain that the "new and protesting offspring" that Doctor Cushing mentioned bringing into the world has been well nourished throughout its pediatric years, survived remarkably well its growing pains, and has emerged strong and healthy into its present maturity at twenty-six years of age.

This, then, is our present status—a large neurosurgical society representing neurosurgery throughout the Americas, and with associations abroad as well—a society that has contributed a great deal to the guidance, growth and stature of neurosurgery over the past quarter century.

Now, we must look ahead and consider our future plans. I believe that most of you will agree that our continued growth is already presenting a problem from the standpoint of future meetings—their location and the physical requirements of hotels such as assembly rooms, dining areas, and the space for exhibitors. There are two questions paramount at this time. First: Can the society reasonably continue with a single-meeting assemblage on an annual basis or will it be desirable to have sectional meetings on a geographical basis? Second: Should we divide the annual meeting into subgroups for special discussion or panel discussions so that a closer participation of the members may be obtained than is now possible in a large assembly? The currently functioning geographically located neurosurgical groups would seem to weigh strongly against any such geographical division of this society. Therefore, it would seem advisable for the Executive Committee and the membership to investigate the possibilities of some changes in the program. These should be designed to further enhance the value of these meetings and to promote closer contact and an exchange of ideas among its members as was originally suggested by the founders of the society.

Further future plans include the enlargement of the Journal of Neurosurgery commensurate with the increase in the material available for publication, traveling fellowships for young neurosurgeons to be sponsored by the society and to be based upon winning essays by the contestants, and contributions to the Harvey Cushing collection of books in the Historical Library of Yale University School of Medicine, in memory of departed members of the Harvey Cushing Society. It will be of great interest to us all to observe the future course of this society and of neurological surgery in the years to come.

The past twenty-five years have witnessed tremendous progress and development in our chosen field. Neurosurgery has come of age in these years, and one can visualize great things for it in the future. I am personally sure that victory over tumors and vascular lesions of the nervous system will be won by the keen minds of our young neurosurgeons who are now embarking upon their careers.

I, like Doctors Sachs, sincerely hope that I may be given the opportunity to see some of these accomplishments fulfilled in the years to come.

I would like to express my sincere appreciation to three good friends who have given me invaluable assistance in the preparation of this address—Dr. Louise Eisenhardt, Dr. Richard U. Light, and Mrs. Donald F. Coburn.

THE ANTERIOR APPROACH FOR REMOVAL OF RUPTURED CERVICAL DISKS*

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(Received for publication September 3, 1957‡)

IN THE surgical treatment of ruptured lumbar intervertebral disks, the operation of vertebral-body fusion following subtotal removal of the disk with partial laminectomy has been employed since 1943.[2] This operation has been performed on over 600 patients and in the writer's experience has proven to be superior to any other operative procedure employed for relief of pain in the back and leg caused by ruptured lumbar disks. The high percentage of permanent cures, the rapid rate of spinal fusion, short period of hospitalization and convalescence, and the early ability of the patient to return to heavy work are the factors upon which these conclusions were drawn.[4]

The poor results obtained in the treatment of disease of intervertebral disks in the cervical region, both from conservative therapy and surgical treatment, has been the concern of surgeons for many years. In an attempt to overcome some of these problems encountered with ruptured cervical disks, the methods used in the treatment of lumbar disks were considered.[3] If the symptomatic lesion of the cervical disk not demonstrated by a myelogram could be identified by diskography and if the pathological joint could be treated surgically by an interbody fusion as is done for lumbar disks, the results from the treatment should be greatly improved. These criteria have been realized by an anterior approach to the cervical disk.

The technical difficulties encountered in the operations currently in use to expose and remove a ruptured cervical disk and/or the osteophytes on the margins of the vertebral bodies may be one reason for the poor results obtained by surgical treatment. The obstacles are primarily anatomical. A comparison between the conventional cervical and lumbar disk operations from a technical standpoint probably explains the excellent results obtained in treating the lumbar disk compared to the poor results for the cervical lesion. The surgical approach to the lumbar disks through the spinal canal is facilitated by the retractability of the long lumbar nerve roots and the dural sac which contains only the cauda equina and cerebrospinal fluid. This approach for performing lumbar vertebral-body fusion is greatly facilitated by use of special self-retaining retractors designed for this operation. With these instruments both the spinal canal and the intervertebral space are

* Presented at the meeting of the Harvey Cushing Society, Washington, D.C. April 22, 1958.
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‡ Revised April 25, 1958.

widely exposed and the danger of injury to structures of the nervous system is minimized. In the cervical region, the protruded intervertebral disk is concealed beneath the large, short, cervical nerve root and/or the spinal cord. The surgical approach used by most surgeons for removal of the disk consists of a complete laminectomy, hemilaminectomy or a facetectomy. The operation is more difficult if the disk has herniated in a dorsolateral position lying entirely beneath the nerve root. There is always danger of injury to the nerve root in obtaining an exposure through the solid bone that overlies it. A Hudson drill or an electrically driven burr is employed for this purpose, as described by Frykholm.[6] The nerve root is also surrounded by large epidural veins, the removal of which may pose a considerable problem in hemostasis before the nerve root can be sufficiently freed and retracted to expose the disk. If the herniated disk or bony osteophytes protrude near the midline, they are exposed by a complete laminectomy. The lesion is removed either by a transdural approach with retraction of the spinal cord,[1] or extradurally working blindly beneath the cord as described by Mayfield.[7] These operative procedures are long and difficult and are frequently fraught with considerable danger and complications. Injury to the nerve root by retraction or hemorrhage may result in serious neurological deficit in the upper extremities. The midline lesions are notorious for the universally poor results obtained by attacking them through the spinal canal.[10]

The anterior approach to the intervertebral disk was suggested while the technique of diskography for lesions of cervical disks was being developed.[5] The radiographic visualization of the intervertebral disk by injecting a radiopaque solution into the disk has long been used to demonstrate the pathological anatomy of the interior of lumbar disks. Lumbar diskography is performed by inserting the needle through the dural sac and into the disk from its posterior surface. The same approach would be impossible for the cervical intervertebral disks since the needle would pass through the spinal cord. The technique of cervical diskography by inserting the needle into the anterior surface of the disk between the carotid sheath and trachea was first performed by Dr. Exum Walker of Atlanta, Georgia and has proven to be a safe, technically simple and yet very valuable diagnostic procedure.[8]

The ease with which the cervical disk could be entered from its anterior surface led to experiments carried out on cadavers to develop a method of removing the disk by this route. A modification of the Wiltberger instruments used in the interbody fusion for the lumbar spine was employed.[9] Wiltberger modified the writer's original operation for interbody fusion of the lumbar spine by designing an instrument which drills a round hole in the intervertebral space into which is inserted a prefit cylindrical dowel of bone. The use of this instrument was found to be an excellent method of exposing the cervical intervertebral disk through the vertebral body (Fig. 1). Accordingly, the instrument was redesigned for this purpose, with an appropriate guard and an adjustable depth gauge. This was necessary to prevent the drill from going too deep, plunging into the spinal canal and damaging the

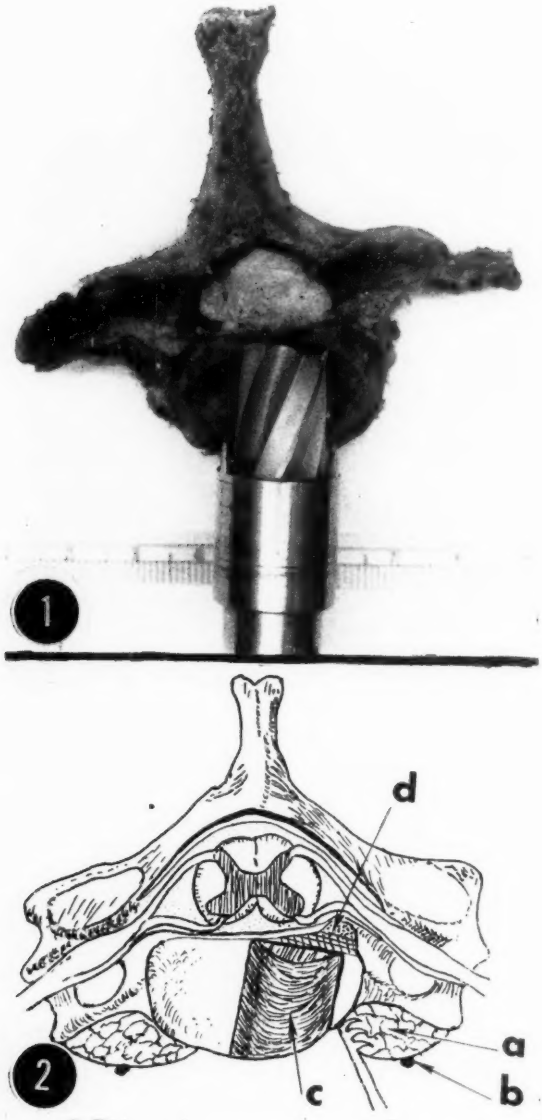

FIG. 1. (1) Photograph of 6th cervical vertebra with drill and guard superimposed on vertebral body. Note width of drill hole compared to spinal canal (actual size).

(2) Drawing to show: (a) longus colli muscle retracted from vertebral body, (b) sympathetic chain, (c) drill hole directed diagonally toward (d) unilateral disk protrusion or osteophyte.

spinal cord. The operation was carried out on several cadavers until the correct instruments had been designed and the technical details of the procedure were perfected. In the past year and a half, 61 patients have been operated upon by this method. This preliminary report will include our first 47 consecutive cases which have been followed from 6 to 18 months postoperative. Several lessons have been learned with this brief experience, through major and minor complications. These are expected, however, in developing a new surgical procedure. This communication will describe the operative technique for removal of ruptured cervical disks and osteophytes by the anterior approach which we have developed and a brief statistical review of the first 47 patients.

OPERATIVE TECHNIQUE

The anesthesia used in the early cases was intravenous Pentothal Sodium with local infiltration in the skin of the neck. An endotracheal tube was used routinely. It was thought that strong retraction of the trachea would induce respiratory difficulties from laryngeal spasm. However, we soon had an opportunity to try the operation on a stoic individual using only novocain in the skin. He suffered no ill effects from the procedure and we were able to stimulate the exposed disk and obtain from the patient valuable objective information as to radiation and location of pain. The operations are now done under local anesthesia with varying amounts of preoperative medication, barbiturates, atropine and an analgesic, depending on the individual patient's needs.

The patient is placed in the supine position with the head turned to the left. The neck is extended over a sand bag to give solid support to the cervical spine. Prior to making the incision, it is necessary to localize by roentgenography the intervertebral space to be operated upon. It is not difficult to make a mistake and choose the wrong disk, as the intervertebral disks all look alike on their anterior surface when exposed at operation. This mistake occurred in the beginning in one patient and led to the following method of preoperative localization. The technique of cervical diskography is employed (Fig. 3B).[5] Digital pressure is made between the carotid sheath and trachea, pressing the finger tips firmly against the anterior surface of the vertebral bodies. A #20 needle is inserted through the skin and down to the bone. Short jabs are made up and down the spine until the intervertebral space is encountered. The needle is then pressed slightly into the disk and a lateral roentgenogram is obtained. If the film demonstrates the needle in the disk to be operated upon, usually C5–6, 0.3 cc. of methylene blue dye is injected into the disk as a marker.

The incision in the skin, 4 to 5 cm. long, is made transversely in one of the natural lines or folds of the neck. The incision extends from the midline to the anterior border of the sternocleidomastoid muscle. For the C5–6 disk, the incision is usually located at the level of the cricoid cartilage. It may be made above or below this level depending upon the disk to be removed, although, as a rule, three disks can easily be exposed through this incision. It is not necessary to extend the incision farther lateral for good exposure. If the incision is made too long, a small sensory nerve (superior cervical), which supplies the skin over the angle of the jaw, may be severed and the patient may complain of numbness in this area after the operation. The skin is dissected subcutaneously and retracted vertically. The platysma muscle is

split longitudinally. Care is taken not to cut this muscle transversely, which assures a much better cosmetic result. The plane of cleavage between the carotid sheath and the lateral border of the thyroid and larynx is easily determined, and the areolar tissue is separated with a blunt pair of scissors down to the anterior surface of the vertebral bodies. Two retractors are used: a superficial self-retaining retractor separates the skin and muscles and soft tissues in a vertical direction, and deep retractor blades are inserted temporarily in the line of cleavage to retract the carotid on one side and the trachea and esophagus on the other. The areolar tissue is cleaned from the anterior surface of the disk. The disk to be removed is identified by the blue dye. The longus colli muscle is cauterized along its medial border on both sides and stripped from the disk and the adjacent margins of the vertebra. The deep retractor blades are inserted into the edge of this muscle on each side. Because the longus colli muscles are firmly attached to the vertebral bodies, strong retraction can be made on them, giving a wide exposure of the disk without applying pressure to the carotid artery or the trachea and esophagus.

The position of the drill hole to be made in the vertebral bodies adjacent to the pathological disk depends upon the site of the suspected disk rupture and/or the osteophytes on the posterior margins of the vertebral bodies as determined clinically. If the symptoms and the cervical diskogram have demonstrated the lesion to be entirely unilateral, the drill hole is placed to that side of the midline. The hole can be placed considerably lateral for excellent exposure of the unilateral protruded disks causing brachial neuralgia and neurological signs in one upper extremity (Fig. 3D). If the lesion, either a disk protrusion or large osteophytes, has been demonstrated by roentgen ray and myelography to occupy a central position, then the drill hole is placed in the midline.

The special instrument for preparing the opening in the bone was designed primarily to prevent injury to the structures within the spinal canal. It consists of a drill and guard which can be used with any Hudson cranial-drill handle (Fig. 2). The guard permits the hole to be drilled to any desired depth. The adjustable screw on the guard allows the drill to advance to within a millimeter of the anterior wall of the spinal canal without fear of injury or damage to structures beyond this point. The guard has small sharp points which are secured to the bodies of the vertebra on each side of the disk by a few light taps of the hammer (Fig.2a). An assistant holds the guard firmly with two hands, the drill is placed inside the guard and the drilling is carried out. The drill and guard are removed two or three times before the desired depth of the hole is reached. This is done to determine the depth and direction of the hole and adjust the length of the guard accordingly, and to remove the soft tissue of the disk. The drill does not remove the soft tissue completely, particularly the cartilaginous plates. The interspace is cleaned out between drillings by using a small sharp curette and a small disk rongeur.

Excessive bleeding from walls of the trephine hole is arrested by rubbing the bleeding bony surface with the bone dust removed by the drill. Bone wax is used in the bottom of the hole but not on the side walls as it would interfere with fusion of the bone graft. The drilling is cautiously continued downward 1 mm. at a time until the bone at the bottom of the hole has a uniform appearance of cortical rather than cancellous bone. This indicates that the cortex of the posterior surface of the vertebral body has been reached. Active bleeding in the bone from subcortical arterioles may occur at this level. This is arrested with bone wax. Removal of the fragments of ruptured disk and the osteophytes on the posterior margins of the vertebral bodies

is then accomplished by using a small, sharp mastoid curette. The bony tissue can be gently separated from the posterior longitudinal ligaments and scooped out in small nibbles, using the upper margin of the drill hole as a fulcrum. A fine pointed, cutting type of disk rongeur will aid in removing soft disk tissue. The curetting process can be carried out on the side of the lesion until the entire posterior and posterolateral margins of the adjacent vertebral bodies have been removed together with the intervening disk. One does not encounter during this procedure vessels of any magnitude. Bleeding from a few small capillaries in the ligament is easily arrested with Gelfoam and thrombin. If the electric cautery is used, it should be

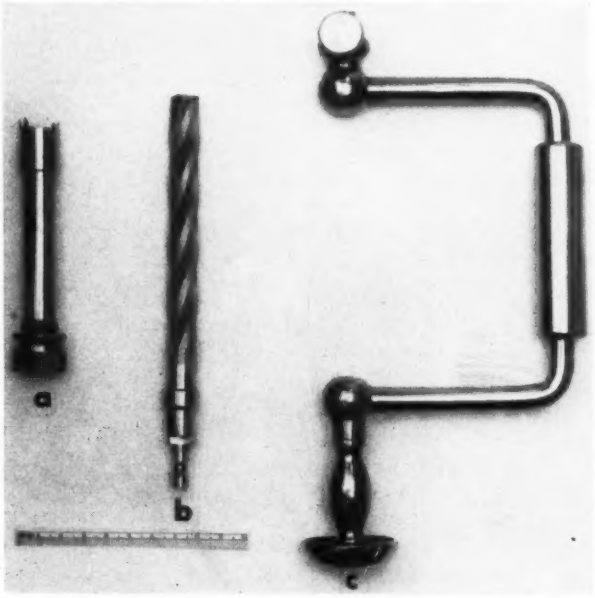

FIG. 2. Instruments used for making the anterior opening into the spinal canal through the intervertebral space. (a) The guard with adjustable screw and lock to vary its length. (b) The twist drill, $\frac{1}{2}$ inch in diameter. (c) Hudson cranial-drill handle.

placed at a low setting because stimulation is transmitted directly to the underlying dura mater and nerve root, and jerking of the arm or body may result.

After the margins of the vertebra and lateral recesses are cleaned of all bone and loose disk fragments, the posterior longitudinal ligament and the posterior fibers of the annulus fibrosus form the covering of the bottom of the drill hole. By careful use of the curette this ligament and its attachments to the vertebra are left intact so that a blind hole results. As stated above, the dura mater and nerve roots and epidural vessels are seldom visualized unless there has been a traumatic rent in the ligament with herniation of the disk material into the spinal canal.

When all bleeding points from bone and ligament are completely arrested by packing the hole with Gelfoam and thrombin, and the hole is dry, the precut dowel of bone is trimmed and placed in the hole and tapped firmly into place with a punch

and hammer. The dowel is carefully measured so that it is slightly shorter than the depth of the drill hole. The dowel is wedge-shaped and 1 mm. or so wider than the hole. By extending the head slightly, a few firm taps on the dowel impact it tightly into the hole. Any oozing from the bone is arrested with bone wax pressed into the groove between the graft and the drill hole.

The bone graft used to fill the drill hole for fusion of the adjacent vertebral bodies is obtained either from the patient or a bone bank. A special instrument, the dowel cutter, removes a cork-like graft by drilling a hole through the patient's anterior-superior ilium. The bone graft can be removed through a 4-cm. incision in the skin prior to the cervical operation. The bone graft was obtained in this manner in only one of our patients, however; in all the others grafts from the bone bank were used. The dowels were made from an ilium removed from a fresh cadaver under sterile conditions, and preserved in blood plasma (for cultures) and frozen.

A small rubber-tissue drain is placed routinely in one corner of the wound down to the vertebra to drain off any blood or serum which may possibly ooze from the bone. A small postoperative hematoma in the wound, although not a serious complication, may cause pain in the shoulder and difficulty in swallowing, and delay healing of the skin. The deep structures of the wound are permitted to fall together and the platysma muscle, subcutaneous layer and the skin are closed in layers with fine silk on an atraumatic needle.

Convalescence from this operation is surprisingly uneventful. Within 24 hours, the patient has recovered from his anesthesia and is able to be up and about. Since no muscles have been incised by the operation, the patient is relatively free of pain. The neck can be moved about freely without limitation or discomfort. A cervical collar or brace was used in the early cases, but this was discontinued. The normal attitude of flexion of the head without a cervical brace is beneficial since flexion compresses the anterior surface of the vertebral body and impacts the bone graft and the vertebral bodies tighter together. This is conducive to early vascularization and fusion. The rubber-tissue drain and sutures are removed in 24 to 48 hours and the patients as a rule are anxious and ready to leave the hospital within 3 or 4 days after the operation. Follow-up roentgenograms of the cervical spine have shown radiographic vascularization of the bone grafts and early fusion of the vertebral bodies within a period of 6 to 8 weeks (Figs. 3C and 4B)!

SUMMARY OF 47 CASES

Within 1 year after this operation was devised, 47 patients were operated upon by this method. There were 29 males and 18 females. Their ages ranged from 33 to 74 years. Half of the patients (24, or 51 per cent) were in the 4th decade. There were 8 patients in their 30's (17 per cent), 12 in their 50's (25.5 per cent) and 2 patients in their 60's (4.2 per cent). One patient was 73 years old. The over-all average age was 46.3 years. Two-thirds (32) of the patients could recall a specific injury to which they attributed their symptoms, whereas 15 patients gave no history of injury of the neck.

The patients were divided into two groups according to symptoms: (a) those with pain only in the neck, shoulder and arm, and (b) those with pain

FIG. 3. *Case 1.* Nurse, aged 46. Pain in neck and shoulder, 8 months. Right brachial neuralgia, 5 months.

(A) Preoperative roentgenogram. Degenerated disk at C5–6 with posterior osteophytes (arrow).

(B) Cervical diskogram showing unilateral protrusion of disk (arrow).

(C) Postoperative film. Disk and marginal osteophytes have been removed and vertebral bodies have been fused. Patient cured.

(D) Anteroposterior film to demonstrate size and location of bone graft (dowel). In this case it was placed off center for removal of unilateral protrusion of disk.

radiating into the forearm, hand and fingers. All 47 patients complained of pain in the neck, shoulder and/or upper arm, and 27 of these had accompanying pain extending into the forearm and hand. There were 20 patients who had no pain or symptoms in the upper extremity indicating nerve-root involvement. All patients whose complaints included pain in the forearm and/or hand with numbness and tingling of the fingers were shown on ex-

amination to have abnormal neurological findings including weakness of the grip, diminution of reflexes and alteration in subjective sensation over the involved dermatome. There were 28 patients with positive neurological findings in the upper extremities and 4 with severe involvement of the pyramidal tract.

Preoperative roentgenograms of the cervical spine disclosed only 6 of the 47 patients to have normal-appearing intervertebral spaces without evidence of osteophyte formation; 3 of these had straightening or reversal of the lordotic cervical curve. In the 41 cases in which there was radiological evidence of degeneration of the cervical disks (Fig. 4A), 47 pathological

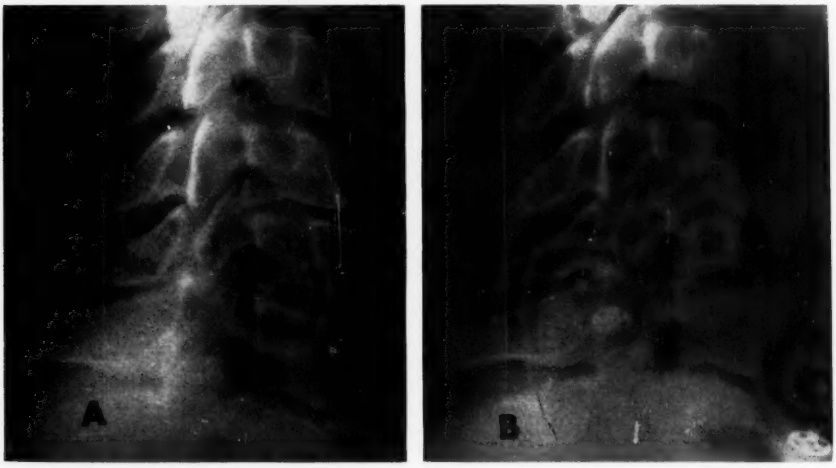

FIG. 4. *Case 2.* Catholic nun, aged 49. "Unbearable" pain at base of neck and shoulders, 3 years.

(A) Preoperative roentgenogram. Severely degenerated disk (cervical spondylosis) limited to a single joint, C5–6.

(B) Postoperative film, 2 months after removal of disk and vertebral-body fusion. Pain completely relieved. (This was the first patient operated upon by this method, before technique for removing posterior osteophytes was developed.)

disks were removed. Twelve of these were considered to be mildly degenerated, 16 moderately and 21 severely degenerated. In the latter group, almost complete obliteration of the intervertebral space with extensive osteophyte formation, either anterior or posterior or both, was present in each case. There appeared to be no relationship, however, between the degenerative changes in the cervical spine, a history of injury and the duration of symptoms. Of the 29 patients with either moderate or marked evidence of degeneration of the cervical disks, 19 gave a specific history of injury to which they attributed their symptoms. In 14 of the 19 patients, the symptoms were of short duration, less than 1 year, whereas the changes in the spine must have been present for a much longer period. Diskograms were performed on all 47 patients and 14 had an additional cervical myelogram.

The pathological disk was demonstrated at the C5–6 level in 31 instances, C6–7 in 9, C4–5 in 4, and C3–4 in 1 patient. Double lesions (two adjacent disks) were found in 6 patients.

The operation for removal of the pathological cervical disks was performed by the anterior approach on 46 patients, and on 1 patient by the posterior approach through a hemilaminectomy. All patients had anterior fusions of the vertebral bodies following removal of the disks. The pathological disk was removed at the C3–4 level in 1 patient (2 per cent). Single lesions were found at C4–5 in 3 patients (6.3 per cent), at C5–6 in 28 patients (59.0 per cent), and at C6–7 in 8 patients (17 per cent). Double disks were operated upon at C4–5, C5–6 levels in 3 patients (6.3 per cent) and 3 others had C5–6, C6–7 lesions (6.3 per cent). In the oldest patient (73-year-old woman) the C4–5 and C5–6 disks were removed and the vertebral bodies were fused. The youngest patient (33-year-old male) had a double fusion (C3–4 and C5–6). This patient had a very short thick neck and because of an error in identifying the correct level, the C3–4 disk was mistakenly operated upon. The error was discovered by roentgenograms 2 days later when the patient still complained of pain in his neck and shoulder. He was returned to the operating room and the correct disk (C5–6) was removed and the vertebral bodies were fused, relieving his pain.

The morbidity and discomfort resulting from this operation is minimal. About the only complaint offered is difficulty in swallowing. This is probably caused by local edema of the esophagus from retraction, for it disappears usually in 48 hours. The pain from the operative wound can be controlled by aspirin. The patient's reaction to any operation for relief of pain can be determined by the length of time he is required to remain in the hospital. Forty of the 47 patients were hospitalized an average of 4.3 days. There were 7 patients who remained in the hospital 10 days or longer (average 17 days) for postoperative convalescence. The longest period of hospitalization was necessitated by the only serious complication encountered with this operation. Because of a technical error in the use of instruments, the posterior longitudinal ligament was separated from the bodies of the vertebrae with subsequent intraspinal hemorrhage and possible direct injury to the spinal cord. This patient was hospitalized for 22 days. The patient had severe neurological deficits immediately following surgery but improved slowly and finally made an excellent recovery with only minimal residual weakness of the right hand and spasticity in the right lower extremity.

The results obtained in this group of patients in relieving pain has been most gratifying. Pain in the neck, shoulder and/or upper extremity was the primary symptom for which the patients sought medical treatment. All patients had received various forms of conservative therapy for months or years without relief. These pains were completely relieved in 42 of 47 patients, and improved in 5. A good example of this group is the oldest patient, aged 73, wife of a doctor, who had suffered pain in the neck, shoulder and upper arms for "many years." She had worn a cervical collar for 4 years and

was never free of pain. Roentgenograms disclosed three degenerated disks (Fig. 5A). At operation the upper two disks were removed under local anesthesia, followed by fusion of the vertebral bodies. The patient remained in the hospital only 2 days. She discarded her brace completely within 2 months. She states the pain she now has is less than 15 per cent of her preoperative pain and is tolerable.

In 8 patients the pain was either not relieved or made worse by the initial operation. These 8 patients were reoperated upon during their initial hos-

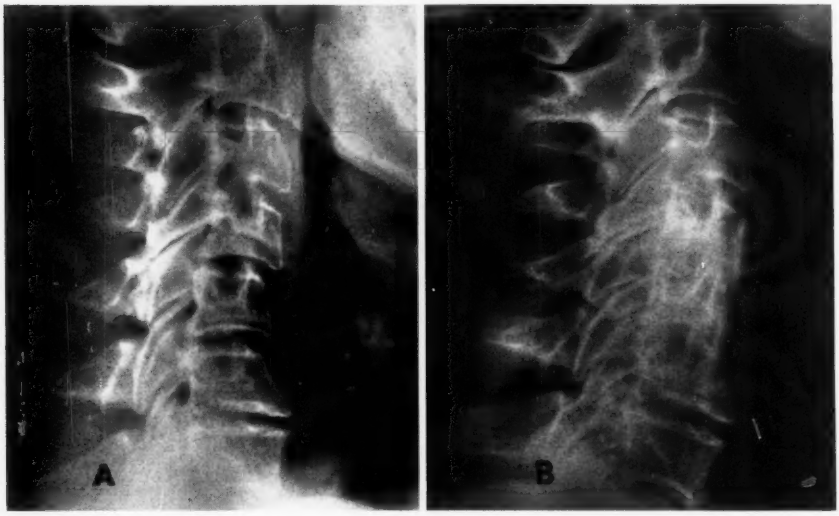

FIG. 5. *Case 3.* Doctor's wife, aged 73. Pain in neck, shoulder and arm "for years". Wore cervical brace 4 years.

(A) Preoperative roentgenogram. Intraspinal midline disk and osteophyte protrusions C4–5, C5–6 and C6–7 (also verified by myelogram). Anterior subluxation C4 on 5.

(B) Postoperative film, 5 months following removal of 4th and 5th cervical discs and fusion of both joints. Patient, 85 per cent relieved of pain; cervical brace discarded.

pitalization or shortly thereafter. In 2 patients a second abnormal disk was removed, followed by fusion of the vertebral bodies. In 4 patients an ill-fitting bone graft was removed and replaced by another. There were 2 patients in whom postoperative hematomas developed. All 8 patients made eventual complete recoveries. There were no wound infections. There were 29 patients who demonstrated neurological deficits preoperatively, including weakness, changes in muscle tone or tendon reflexes, and demonstrable sensory change with subjective numbness and paresthesias. A complete recovery occurred in 20 of these patients, 6 were markedly improved and the symptoms remained the same in 2. One patient was considered worse.

In this series are included 3 patients with acute traumatic laceration of the disk and fracture dislocation of the cervical spine. A detailed description

of these cases is the subject of another report. Suffice it to say that removal of the injured disk and vertebral-body fusion by this method performed shortly after injury have shortened the convalescent period of these patients 3 to 8 weeks.

Postoperative roentgenograms have been obtained at frequent intervals to determine the fate of the bone grafts and the rate of fusion. Fusion occurred in 6 to 8 weeks in the majority of the cases. A complete interbody fusion occurred in 44 patients within 3–4 months after operation. In 3 patients, the bone graft underwent absorption. One of these patients is pain-free; the other 2 have tolerable residual pain in the neck and shoulder.

COMMENT

This operation is indeed a most welcome and gratifying substitute for the conventional methods of surgical attack on ruptured cervical intervertebral disks through the lamina and spinal canal. The operation is a great deal more simple. From the patient's standpoint, the operation is considerably less painful and disabling than other surgical procedures, with rapid postoperative recovery and short period of hospitalization. Technically, it is possible to remove completely both soft-tissue and bony elements encroaching upon the nerve roots or the spinal cord a great deal more satisfactorily than has ever been possible by the posterior approach. The dangers of injury to the spinal cord and nerve roots in the posterior operation, usually by hemorrhage and/or retraction, are almost entirely eliminated. Finally, the most important contribution to the treatment of degenerated cervical disks (cervical spondylosis) by this new procedure is the fact that the vertebral bodies are firmly fused together in a very short period of time. This arrests the degenerative process at the joint which, if permitted to continue, may accumulate large osteophytes on the margin of the vertebra with encroachment upon the spinal canal and possibly result in chronic irreparable damage to the spinal canal and/or nerve root.

A discussion of the criteria and indications for this operation has not been included in this presentation. Likewise a description of pain resulting from disease of the cervical disks has been omitted. Our experience with this surgical approach has presented a singular opportunity to study the problems of pain encountered with these lesions. The data which have been accumulated are most enlightening and will be reported in a later communication.

SUMMARY AND CONCLUSIONS

The operative technique for removal of ruptured cervical intervertebral disks by an anterior approach followed by vertebral-body fusion is given in detail. The rationale of this operation and the advantages over the conventional surgical methods of treating cervical disks are presented. The operation is not technically difficult. As far as the patient is concerned, it may be classified as a minor operation resulting in very little if any postoperative pain or complication and requiring only 3 to 4 days of hospitalization. For this rea-

son, therefore, one may be justified in offering this type of treatment to patients with disease of the cervical disks in lieu of the so-called "conservative treatment" consisting of traction of the head, analgesics, cervical braces, heat treatments, etc. These forms of therapy frequently are ineffectual in relieving the patient's clinical symptoms, even when administered over a period of weeks or months. Although our experience with this operation is limited, the gratifying results obtained in relieving pain in the neck, shoulder and arm would indicate a bright future for this form of surgical treatment. The anterior surgical approach may replace some operative procedures now employed for treatment of clinical symptoms resulting from rupture and degeneration of the cervical intervertebral disks.

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DISCUSSION*

Dr. John Raaf: Your Program Chairman, Dr. Jack French, asked me to substitute for Drs. Spurling and Mack in discussing these papers. I realize that neither mentally nor physically can I fill the shoes of those gentlemen. After Dr. French made his request, I searched my hotel room for material for the discussion. I could find only the Gideon Bible. If my remarks seem a little old-fashioned, it may be because of the fact that my source material is not quite as up-to-date as that just presented.

For some time I have been trying to decide whether anterior cervical fusion is in the same category as Russian tonsillectomy. Perhaps you all saw the report from the Soviet Union a few years ago that tonsillectomy is performed from the lower end of the alimentary canal because the Russian citizen is not allowed to open his mouth.

I am a great admirer of Dr. Cloward's prodigious energy and his abundance of ideas but so far I have not been able to bring myself to do either discograms or anterior cervical fusions. It is true that there are a number of very fine blind pianists, but I doubt that one can pound on the strings that make up the brachial plexus with the same abandon that one can pound on piano strings.

One reason I have avoided discograms is the pain involved. Some patients I have seen

* In addition to Dr. Cloward's paper, another on the same subject was presented at this meeting by Drs. G. W. Smith and R. A. Robinson (see *J. Bone Jt. Surg.*, 1958, *40-A:* 607–623).

after discograms have been performed by others, have informed me it was necessary for them to stay in bed 2 or 3 weeks after the test because of pain. I wonder if Dr. Cloward would comment on this point?

If anterior cervical fusion has merit, I think the indications should be clearly delineated. Sometimes, regardless of what one does to a patient's spine, he improves anyway. I recall that 8 or 9 years ago I performed a myelogram and visualized a large cervical protruded disc. Because there was a common spinous process for the 2nd and 3rd cervical vertebrae, I miscounted and explored the wrong space. The patient recovered and worked comfortably for years until recently he fell and again had cervical disc symptoms. At an operation a short time ago I removed the protruded disc which had been visualized 8 or 9 years previously.

Dr. William B. Scoville: I have been asked to give the "Hartford" approach to this problem. For some years, we have offered an exhibit on cervical discs which is presented again at this meeting, representing the work of the speaker and associates, Drs. Whitcomb, Dunsmore and Reilly, surveyed by our former resident, Dr. Robert McLaurin. This exhibit is based on 400 operative cases. In discussing the interesting papers by Dr. Cloward and Dr. Smith, I ask that we define our terms of classification because of the existence of four really distinctly different forms of cervical ruptured discs: (1) The central soft disc, a very rare and very serious condition generally causing paraplegia. (2) The central spondylosis or bony-ridge type which gives a pseudo-amyotrophic lateral sclerosis picture of anterior root plus long tract signs, a chronic and not common syndrome. (3) The laterally placed cervical discs consisting of the bony, hard, or osteophytic type, causing a chronic, nagging, low-grade discomfort in neck and arm. (4) The true "soft" lateral cervical ruptured disc which may be chronic or acute and utterly or mildly disabling.

In our 400 operative cases, 388 were lateral, only 12 were central, and 340 of the lateral ones were of the soft variety. Hence, the bulk of our experience is with this latter type, and in this type I presume to take issue on the necessity for an anterior approach. My friend, Rupert Raney, originally presented the anterior approach to the laterally placed bony type of cervical ruptured disc at a meeting of the American Academy of Neurological Surgery some 10 years ago. He has given up this approach because he feels the posterior approach through the facet is simpler and because of one serious mishap attributed to thrombosis of the anterior spinal artery. We tend towards this feeling of a simpler approach because of the extraordinarily good and rapid results obtained in our keyhole, posterior exposure through the facets with uncapping and unwalling of the involved root by the use of an electric drill and a milking out of the soft disc fragment without curettage of the interspace. So far, there have been *no* recurrences in contradistinction to the lumbar discs. In the case of the osteophyte or bony type, decompression alone by the same technique is generally sufficient. There has been zero mortality, zero infection, and a review of the results in 165 cases of industrial workers showed that 97 per cent were improved, with 86 per cent markedly improved, and with all but 3 returning to work at their former jobs. Sixteen per cent returned to work in under 2 weeks and 60 per cent in under 1 month. The only appreciable morbidity was 2 cases of complications attending local block anesthesia and 3 cases of air emboli, two of them grave, which we believe can be forestalled with adequate precautions and should always be guarded against when the patient is in the upright position.

Aside from the simplicity and excellence of results in the posterior keyhole, facet, electric-drill approach, mention should also be made of an observation which may militate against the anterior fusion approach. That is, approximately one-fourth of our laterally placed ruptured soft discs have shown the rupture to be at the interspace adjacent to but not at an interspace revealing arthritic narrowing and partial fusion by roentgen ray. This suggests to us that a damaged or partially immobilized interspace puts additional strain on the space above or below it, resulting in lateral rupture of the disc. May not fusion of a cervical interspace cause this same stress on adjacent interspaces?

I wish to thank you for this opportunity to discuss this stimulating subject.

DR. A. EARL WALKER: When Dr. Smith first attempted this procedure some 5 years ago, I thought it was a logical approach to some of the problems of cervical disc, and when he left Hopkins we have continued, with Dr. Robinson, to carry out this procedure.

We did find that some of our patients complained of discography, so that recently we have been carrying this out at the time of operation. This has the disadvantage in that the patient can't tell you when his pain is reproduced. The site of the degenerated disc can usually be ascertained by the dispersion of the dye.

We have also modified the procedure a little bit in that we now put the head in traction to open up the intervertebral space when the disc has been removed, which I think allows less likelihood of compression of the root posteriorly by spurs which may be left.

I would affirm the statements that these patients can be gotten up in a few days. Many will go back to work in a couple of weeks.

DR. GEORGE PERRET: We first heard of this procedure in 1954 when Dr. Dereymaeker, of Louvain, Belgium, traveled in this country and visited various neurosurgical centers, reporting on 34 cases that he already had at that time. He demonstrated his operation to us and I understand also in other neurosurgical clinics; his method of operating was exactly the one that was shown by Dr. Smith today.

He reported success in treating root irritation and also long tract signs in cases of discs, cervical ridge syndrome and other types of cervical arthropathies. Since that time we also have done this operation with satisfactory results.

DR. RALPH B. CLOWARD: One always encounters objections when presenting something new and out of the ordinary. The old statement that "you can't teach an old dog new tricks" may be applicable to this new cervical disk operation. Since most of us are reluctant to give up a surgical procedure, that has proven to be satisfactory, for something new, Dr. Raaf is perfectly justified in his objections. I know the difficulty he encounters in his state with industrial accident cases, labor unions and lawyers processing litigation cases.

I might mention here that none of the patients in this series has had law suits pending for whiplash injuries caused by automobile accidents or other mishaps. They are patients who have suffered persistent disabling pain in the neck, arm and shoulder for months or years: pain that has not responded to any form of conservative treatment. In this group of patients, the results obtained in relieving the pain by a relatively minor surgical procedure have been so spectacular we feel the operation is justified.

The question about pain from diskography: The manner in which the diskogram is performed will determine the amount of pain the patient will experience.

The cervical disc is a relatively small structure. The nucleus pulposus of a normal cervical disc will accept only 0.2 to 0.3 cc. of a liquid solution. Using the 2-needle technique, a very fine needle is inserted into the nucleus and injected with a tuberculin syringe. If the disc is normal and only 0.3 cc. is injected, the patient experiences little or no pain. When a pathological disc is injected the pain may be intense, but lasts only from 3 to 5 minutes. The patient will accurately localize the pain, which is often identical with his clinical symptoms. The pain usually has completely disappeared by the time the patient leaves the x-ray room. I have never encountered a patient who complained of persistent pain in the neck or back following a cervical or lumbar diskogram which lasted more than 24 hours.

Dr. Raaf's question as to the indications for operation, I believe has been answered. My patients have had chronic lesions which had not responded to other forms of treatment, and they have no legal suits pending. This is the type of case that should be chosen. Dr. Scoville does a different type of operation depending on which of the four varieties of disc he is treating. With this operation, it makes no difference what type of disc the patient has or its location. Whether it is a soft disc or a hard one, whether it is located in the midline or to one side, they can all be rapidly and safely removed by a single anterior midline approach. With the posterior method, one may remove one lesion and leave another, that is, remove a small fragment from beneath the nerve root and leave the large midline osteophyte. This leaves the

patient with the possibility of symptoms developing at a later date or continuing with symptoms in the neck or shoulder. By this new operation all pathology is removed and the joint is immobilized, thus eliminating all conditions that can be responsible for pain.

The simplicity of this operation from both the surgeon's standpoint and the patient's is most gratifying. From the surgical standpoint, there is no comparison. We have all struggled with laminectomies, drilled holes in facets, spent hours trying to arrest bleeding from large epidural veins; and then struggling to remove a large hard lump from beneath the tight swollen nerve root. If a lesion is midline an extensive laminectomy with retraction of the cord is necessary. The dangers to the nerve roots and/or the spinal cord by these procedures are obvious.

By this operation, there is no harm done to the nerve roots or cord because these structures are never seen or handled. That is the simplicity of the procedure from the surgeon's standpoint.

From the patient's standpoint, I have had patients tell me the operation was not as bad as a tonsillectomy, be it Russian or otherwise, because they experienced little or no pain from the procedure. The only thing that is cut with the operation is the skin. Since no muscles have been cut the patients can move head and neck around freely and can be up and about the following day and they are very happy.

CEREBRAL ANGIOGRAPHY: A NEW TECHNIQUE

CATHETERIZATION OF THE COMMON CAROTID ARTERY VIA THE SUPERFICIAL TEMPORAL ARTERY

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(Received for publication September 9, 1957)

METHODS of catheterization of peripheral arteries have been developed in recent years and applied to both physiologic studies[4,7,8,11] and intra-arterial injection of drugs. Fractionated administration of nitrogen mustards into the external carotid system via a plastic catheter introduced through a peripheral artery has been used as a palliative measure in the treatment of malignancies of the head and neck.[2,6,13] It seemed to us, therefore, that a satisfactory application of such a technique to the internal or common carotid artery would offer a method of cerebral angiography devoid of the hazards of direct trauma to the carotid arteries and adjacent structures incident to presently employed percutaneous and open techniques.

Catheterization techniques have been applied to cerebral angiography in several ways. Radner[9] has developed a method of vertebral angiography in which, under fluoroscopic control, a catheter is passed up the radial artery to the vertebral artery. It is a complicated technique and, to our knowledge, has not been applied to carotid angiography. Jaeger and Whitely,[5] in 1955, reported an intravascular intubation technique in which a plastic catheter is introduced into the carotid or vertebral artery through a needle placed percutaneously into the appropriate vessel. These authors point out the advantages of their method: (1) elimination of the danger of extravascular injection, thus making Thorotrast a more desirable medium, (2) prevention of mechanical damage to the intima of the artery with reduction of complications related thereto, (3) precise injection of the internal carotid artery is possible without puncturing that vessel, (4) elimination of the strain of maintaining a sharp needle immobile in the artery, (5) freedom for proper positioning, and (6) allowance for a leisurely, accurate study. The chief disadvantages of their technique would seem to be: (1) the necessity for special equipment, not readily available, and (2) the continued necessity of the trauma and sequelae incident to puncture of a major artery. We believe our method combines the essential advantages outlined by these authors and eliminates the disadvantages.

The superficial temporal artery was selected as the vessel of entry into

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the carotid system because of its size, accessibility, relatively straight course, and because it can be sacrificed with impunity. The external carotid artery terminates between the upper part of the parotid gland and the back of the neck of the mandible by giving off the internal maxillary artery and continuing in almost a linear fashion as the superficial temporal artery which emerges from the parotid gland immediately inferior to the zygoma. Over the zygomatic process and in front of the auricle it is quite superficial, being covered only by skin and fascia and can easily be felt pulsating. In this region it is accompanied by the auriculotemporal nerve, which is posterior to it,

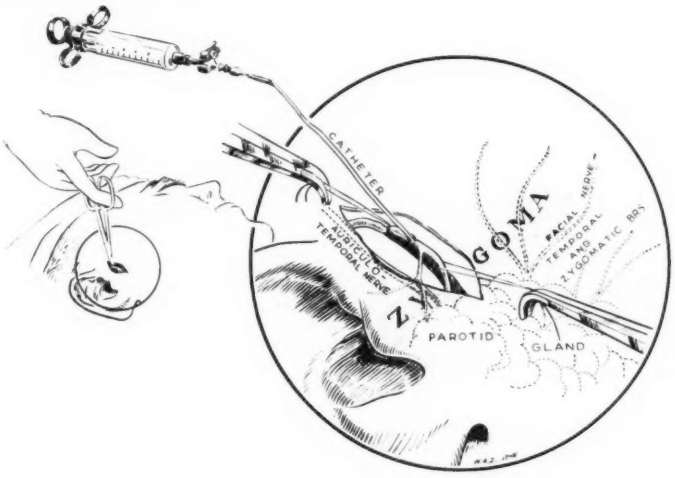

FIG. 1. Drawing of incision utilized. The enlarged inset shows the relations of the superficial temporal artery to its vein, the facial nerve, parotid gland, and zygoma.

and by its vein which has no constant position, but is often superficial to it. Temporal and zygomatic branches of the facial nerve also emerge from the upper border of the parotid gland, but are well anterior to the artery (Fig. 1). Approximately 2–5 cm. above the zygoma, the artery ends by dividing into frontal and parietal branches.

TECHNIQUE

Because of certain difficulties encountered in the development of this method, we feel it is important to describe in some detail the techniques employed.

Only a small area in the lower temporal region need be shaved. Under local anesthesia, a vertical incision in the skin, approximately 2 cm. long, is made in the preauricular region over the palpable temporal artery. The lower end of the incision extends to the lower border of the zygoma. The edges of the skin are slightly undermined and small hemostats are applied to the subcutaneous tissue for retraction. Fascia is thus exposed under which the artery can be seen or felt pulsating. It is

isolated by blunt dissection and exposed in the extent of the incision. The vein should not be mistaken for the artery. The latter is quite redundant when exposed and visibly twitches with each systole. The vein occasionally requires ligation and division if it overlies the artery. The artery is then ligated distally, using 3–0 plain catgut, and the uncut ligature is grasped with a hemostat for traction. A similar suture is passed under the vessel proximally, left untied, and also grasped with a hemostat for traction.

For the plastic catheter we have found it convenient to use Clay-Adams Intramedic No. PE 190/S12, which is supplied in individual sterile packages in 30 cm. lengths (inside diameter .047 inches, outside diameter .067 inches) and is usually stocked by hospitals for use in venous cut-downs. A blunted 18 gauge needle fits snugly into one end and is connected by a stopcock to a syringe of saline. The other end of the tubing is cut on a bevel to facilitate entry into the vessel.

The exposed artery is then lifted between the two sutures and gentle traction is maintained. A small nick is made into the artery and the beveled end of the catheter is inserted. Once into the lumen, traction on the proximal suture is relaxed, and the catheter is advanced inferiorly 12–15 cm. using the distal suture for countertraction. This distance will place the catheter into the common carotid artery. The proximal suture, previously left untied, is now tied to secure the catheter in place. An additional catgut ligature, to be tied when the catheter is withdrawn, is looped around the vessel and catheter. The skin incision is then closed around the catheter. A temporary dressing is applied and injections of a suitable radiographic medium are made in the conventional manner. We have used 50 per cent Hypaque, 6–8 cc. for the lateral view, and 8–10 cc. for the axial views. Between injections, the catheter is irrigated with saline. At the conclusion of the procedure, the catheter is slowly withdrawn and the vessel is ligated proximally. Should the ligature slip, adequate hemostasis can readily be secured by firm pressure applied for several minutes.

The chief problem encountered has been the meeting of obstructions to the advancement of the catheter immediately after entering the vessel where the vessel forms almost a right angle rounding the zygoma. This can be avoided by entering the artery as low as possible and by directing the catheter perpendicular to the surface of the skin after entry. Should obstruction be met, it can usually be overcome by slow advancement and rotation of the catheter and simultaneous injection of saline. The return of arterial blood into the catheter should be checked frequently if there is difficulty. On one occasion, we were unable to pass the catheter more than 5 cm. because, we thought, of impingement of the catheter at the point of origin of the internal maxillary artery. Nevertheless, a satisfactory angiogram was obtained by retrograde flow of the contrast medium into the common carotid artery.

RESULTS

Using the technique outlined above, we have performed 65 successful carotid angiograms in 48 patients in whom angiography was indicated for diagnostic purposes. Early in the study, we experienced trouble overcoming the obstruction associated with the acute turn in the vessel around the zygoma and because of this, on four occasions, the attempt to catheterize the

vessel was unsuccessful. Since recognizing the cause of the obstruction, the importance of exposing the artery low in the region of the turn and of directing the catheter inwards when this point is reached, we have had only occasional difficulty threading the catheter the desired distance and no failures.

With the catheter threaded down 12–15 cm., we have had uniformly excellent results from the radiographic standpoint. In all cases, adequate filling of the internal carotid system was noted and excellent technical studies resulted (Fig. 2).

FIG. 2. Routine lateral and cross-fill anteroposterior views using this technique. The dye-filled catheter can be seen across the skull in the lateral view.

The fact that the injection is made retrograde to the blood flow has not diminished the contrast obtained, nor has the point at which the exposure is called for during the injection required a change in timing.

No complication has occurred as a result of ligation of the temporal artery. Six patients have had scalp flaps heal uneventfully, 3 following unilateral ligation, and 3 following bilateral ligation. No injuries occurred to the facial nerve or its branches, nor have there been any complications referable to the parotid gland. Following the procedure, one patient noted hypesthesia in the distribution of the auriculotemporal nerve which spontaneously cleared. One major complication occurred in this series. We feel that it is important to present this case in some detail because we do not believe the complication is attributable to the method per se, but is rather a complication of angiography in general. Indeed, it is possible that the symptoms observed represented the natural progression of the disease process, not ascribable to angiography.

CASE REPORT

A 40-year-old pilot was well until 1 month prior to admission, when he suddenly fell to the floor, became disoriented, and was found to have a left hemiplegia. He complained of moderate right-sided headache. He was taken immediately to the local base hospital, where, on admission, he was found to be slightly confused and presented a complete profound left hemiplegia, including face, tongue, arm, and leg. The eyes were deviated to the right and the right pupil was slightly larger than the left, but both reacted well. Mild hypalgesia and hypesthesia were also present over the entire left side. The white blood count was 11,450 per c.mm. with a normal differential, hemoglobin 14.5 gm. per cent, nonprotein nitrogen 33.3 mg. per cent and glucose 94 mg. per cent. Lumbar puncture revealed clear cerebrospinal fluid. The initial pressure was 220 mm. of cerebrospinal fluid, cell count 16 lymphocytes per c.mm. and protein 35 mg. per cent. Roentgenograms of the skull and chest and an electrocardiogram were normal. Over a 2-hour period, without specific treatment, there was complete recovery. He was transferred to this hospital for further evaluation. The past history and system review were negative.

Physical and detailed neurologic findings upon admission to this hospital were entirely within normal limits. The routine laboratory studies, including serology, revealed no abnormalities. The visual fields by perimetry and tangent screen were normal. An electroencephalogram was normal. A lumbar puncture was repeated and yielded clear, colorless cerebrospinal fluid under pressure of 120 mm. of cerebrospinal fluid; cell count was 7 lymphocytes per c.mm., protein 36 mg. per cent and serology was negative.

Two months after his onset of symptoms, right carotid arteriography was performed under local anesthesia through a catheter introduced through the right superficial temporal artery a distance of 15 cm. A total dosage of 38 cc. of 50 per cent Hypaque was employed. Narrowing of the right anterior cerebral artery distal to the anterior communicating artery was noted in all views; the study was otherwise normal.

He tolerated the procedure well and was asymptomatic until 23 hours following the completion of the angiogram. At this time, he arose from bed and while walking in the ward, suddenly became disoriented and complained of nausea. When seen shortly thereafter by the doctor, he was again hemiplegic on the left, the eyes were deviated to the right, and a profound left hemihypesthesia was present. He was treated with immediate and repeated stellate blocks without dramatic effect, although there was moderate improvement over the next 24 hours. Improvement continued slowly thereafter until 1 week later when he awoke from sleep with increased weakness, most marked in the left leg and moderate in the arm and face. Again he was somewhat disoriented and confused, but this cleared rapidly over an 8-hour period. The hemiplegia slowly improved thereafter, but residual weakness was still apparent 2 months later.

DISCUSSION

The complications of angiography can be attributed to two major factors: first, the passage of the dye, a foreign substance, through the cerebrovascular system, and second, the trauma incident to the introduction of the material into the vascular system. Obviously, the reduction of complications caused by the first factor—the effect of the dye—will depend fundamentally on the

development of better media, as well as careful management of the procedure, such as minimizing the number of injections, the volume of each injection, and insuring an adequate interval between injections. The presently reported technique should not affect the incidence of complications caused by this factor. It should, however, facilitate the management of the procedure insofar as permitting an adequate interval between injections without fear of dislodgment of the needle.

The second factor, the trauma incident to needle puncture, has received increasing attention in recent years. Crawford[3] studied lesions resulting from arterial puncture in necropsy material from 75 patients dying at intervals varying from a few hours up to 8 months after cerebral arteriography. His material did not lend itself to statistical analysis, but emphasizes the pathologic effects of vascular puncture. His findings included (1) bleeding in and around the carotid sheath, (2) sterile abscess formation in the adventitia and sheath of the artery, (3) thrombosis, both occlusive and mural, (4) cerebral embolism, and (5) aneurysm formation, both traumatic false and traumatic dissecting. Rowbotham *et al.*[10] performed autopsy in 6 cases within 10 days of angiography and found hemorrhage in the periarterial tissues in all cases, in none of which it was felt to be clinically significant. The intima of the posterior wall of all the carotid vessels punctured had received one or more lacerations up to 3 mm. in length. In 1 case, the laceration was associated with a friable mural thrombus. Sirois *et al.*[12] reported an interesting case with necropsy confirmation in which the intima of the posterior wall of the common carotid artery was lacerated to form a valve-like flap which obstructed the cephalad flow of dye.

The importance of these pathologic findings in the production of clinically manifested effects is difficult to assess. This is in part because of the lack of description of the postmortem state of the vessels of the neck in fatal complications reported previously. Even when pathologic effects of vascular puncture are evident, it may be difficult to relate them to the complication observed. It is likely that a clinical study of statistical significance in which the factor of needle puncture has been eliminated will be required to properly evaluate the role of trauma in the production of complications. Our technique offers a method for such a clinical study.

The group of extravascular complications directly attributable to trauma and extravasation of dye are eliminated by this technique. This group includes irritation of cervical soft tissues, damage to the vagus and cervical sympathetic chain,[1] and brachial plexus neuritis. These later complications do not contribute to the mortality rate, but do increase the morbidity rate of the procedure.

We have been pleased with the freedom of positioning of the patient available to us with the catheter in place as compared with the situation with a needle in place. Patients can be positioned accurately on the roentgen-ray table without fear of vascular trauma or dislodgment of the needle. Since the intracranial carotid system lies more anteriorly than posteriorly,

increased definition of this system should be obtained in posteroanterior projections rather than anteroposterior projections. When indicated we have employed posteroanterior projections including straight posteroanterior, optic foramen, and other oblique views. In routine roentgenography the view of the optic foramen is taken in the posteroanterior position since this places the orbit close to the film and allows for maximum detail. We have found that this principle applies to angiography as well, giving maximum detail of the vasculature related to the optic foramen and orbit (Fig. 3).

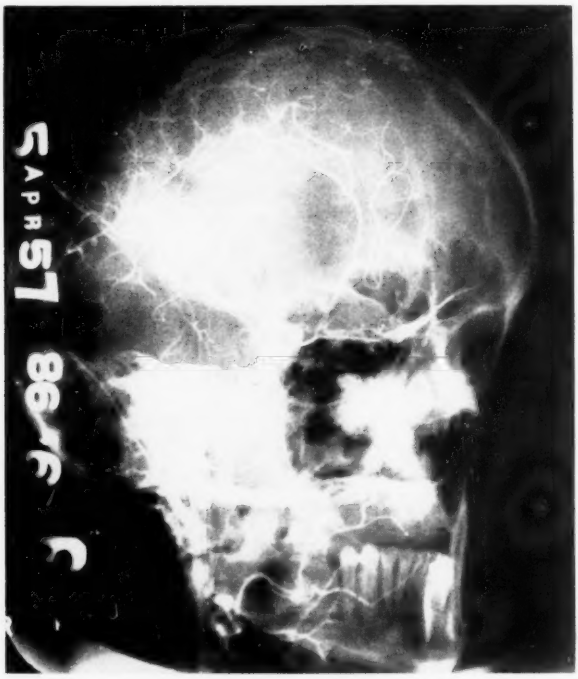

FIG. 3. Posteroanterior view of optic foramen using this technique.

When an oblique view of the carotid system is indicated, we have found the posteroanterior projection superior to the conventional anteroposterior views. In all posteroanterior views the vessels as outlined by dye are smaller, denser, and more sharply defined than in comparable anteroposterior views.

All patients were operated upon under local anesthesia, which we prefer. This included several patients who were quite apprehensive, several who were irrational, and thus uncooperative, and in whom percutaneous angiography would have required a general anesthetic. If the operator prefers general anesthesia, intratracheal intubation is not required since there is no

danger of tracheal compression and since the airway is readily accessible at all times. Another advantage is that this procedure is relatively benign insofar as the patient is concerned. Several of the patients had angiography performed both by percutaneous and catheter methods. These patients preferred the intubation method because of increased comfort during and after the procedure. They are not required to lie immobile on the table during the entire procedure, and following the procedure, they are invariably more comfortable than after injection of the neck.

Other advantages of more limited application are the feasibility of using Thorotrast without concern for extravasation and the ability to visualize the external carotid system. The study of this system may be desired for additional information in certain cases of neoplasms, vascular malformations and occlusions.

The disadvantages of our method are apparent. It is a more complicated procedure and takes somewhat longer to do than the percutaneous method in skilled hands. However, the surgery is simple compared to exposure of the carotid itself. Since perfecting the technique, the average time required for the surgery involved has been 20 minutes. The procedure can be carried out on the roentgen-ray table and the instruments required are those that are readily available in the usual venous cut-down set. A second disadvantage is that it is an open technique and the possibility of infection of the wound exists, but has not occurred in our series. A facial scar results, but because the incision lies along the normal lines of the skin and lies in part within the hairline, an excellent cosmetic result is obtained. The fact that the external carotid system is filled routinely may be considered a disadvantage. It is possible that ligation of the superficial temporal artery may result in vascular insufficiency in a related scalp flap, although this has not occurred in our limited series. A distinct disadvantage is that the method entails sacrifice of the artery, and is therefore not available for use in repeated studies.

Other possible applications of this technique under consideration include retrograde vertebral angiography, cerebral angiography in children, subclavian angiography, and aortography. We have begun to explore the possibilities of obtaining vertebral angiograms by threading the catheter down the right side into the innominate artery. Pressure is applied to the ipsilateral carotid and a sphygmomanometer cuff is inflated on the right arm to obtain retrograde vertebral filling. Thus far, we have not been consistent with our vertebral filling. More data are required with respect to the necessary volume of dye and the exact timing, but the preliminary studies are promising. Carotid studies by this technique have been carried out successfully in 1 infant, aged 10 months.* The temporal artery is large enough for catheterization, though perhaps a smaller tube would be required, as in the infant above, in which a #90 catheter with a 21 gauge needle was em-

* A patient of Dr. Robert M. N. Crosby, Baltimore, Maryland.

ployed. The normal and pathologic angiographic anatomy of the subclavian artery may prove to be a practical aid in the evaluation of thoracic outlet syndromes.

SUMMARY

1. A new technique of cerebral angiography has been described which utilizes catheterization of the carotid system via the superficial temporal artery.

2. The results obtained by this technique in 65 angiograms done on 48 patients have been presented.

3. This method essentially eliminates trauma caused by puncture of the neck, and therefore lends itself to the study of the role of such trauma in the genesis of complications attributable to angiography.

4. The advantages and disadvantages of this method have been discussed.

5. Further application of this technique to vertebral angiography, cerebral angiography in children, subclavian angiography, and aortography has been outlined.

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THE FREQUENCY OF INTRACRANIAL AND INTRASPINAL NEOPLASMS IN THE RESIDENT POPULATION OF ROCHESTER, MINNESOTA

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(Received for publication September 19, 1957)

The purpose of this paper is to report the incidence, prevalence, and death ratio for various neoplasms affecting the central nervous system in the resident population of Rochester, Minnesota, for the 10-year period 1945 to 1954, inclusive.

It is desirable to have such statistics for various populations. Such information might disclose differences in frequency by race, geographic area or characteristics of population and would be of value in planning for research and needs of medical care. Epidemiologic studies in areas with appreciable variances in incidence might provide relevant clues to etiology.

Most previous reports dealing with the frequency of neoplasms affecting the central nervous system have been based on the personal experience of practising neurosurgeons or upon selected necropsy series or proportionate rates of hospital admissions. These data, although of value in describing clinical or pathologic experiences, do not necessarily provide a true picture of the incidence of neoplasms or of their frequency by type in the total population.

Surgical series are more likely to be representative of operable neoplasms rather than of all neoplasms in the population; operability, in turn, is influenced by such characteristics as age and general health of the patient and by the presumed type or accessibility of the lesion. The interest and reputation of the surgeon may influence the nature of his series; the relatively high proportion of pituitary tumors in Cushing's series[1] of intracranial neoplasms is an often cited example of this type of bias. The admission policy of a hospital, such as age, sex or racial limitations, or its emphasis on neurosurgical problems may present another form of bias that prevents accurate generalizations from its data.

Series of cases seen at necropsy may likewise be unrepresentative because of the frequency of special interests in research in neoplastic conditions and because of the greater effort expended by the resident staff in obtaining permission for necropsy in cases of puzzling or relatively uncom-

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mon disorders. The rate of 2 per cent mentioned by Merritt[9] as the proportionate rate of necropsy for brain tumors contrasts with the death ratio of 0.5 per cent of the deaths in the United States for the years 1952 to 1954, inclusive, that were certified as caused by neoplasms of the nervous system.[12]

Recently a series of monographs on the incidence of cancer in specified populations has been published describing the results of extensive surveys in 10 metropolitan areas in the United States and in urban and rural Iowa. In these studies, an attempt was made to register all diagnosed cases of cancer, including neoplasms of the central nervous system, for a preceding calendar year.[2,5] Similar data were obtained for Connecticut[4] in a long-term study of cancer-registry reports. Comparisons have been made of the cancer rates, geographically, by age, sex and race; some of the results of these studies relating to the central nervous system will be compared with the results in Rochester, Minnesota, to be presented here.

Most surveys dealing with tumors of the central nervous system are limited to primary neoplasms. In a few instances, however, data have been presented on the relative frequency of primary and metastatic neoplasms observed at operation or necropsy. Since operations on the brain or cord are less likely to be undertaken in the face of recognized metastatic disease, the metastatic lesions are probably underestimated in the surgical series.

The Selection of Rochester, Minnesota. It is the impression of the author that the excellent and in many respects unique facilities for medical care* concentrated within the organization of the Mayo Clinic are able to provide unusually complete statistics for many serious disorders including neoplasms of the central nervous system for the community's 30,000 residents. A substantial proportion of the local residents are examined during life at one of the facilities of the Mayo Clinic or after death by the pathologists of the clinic.

It is assumed in this study that all residents of the community in whom neoplasm of the central nervous system was suspected or diagnosed during 1945 to 1954, inclusive, were observed at one of the facilities of the clinic and that data on these patients were included in the clinic's unified record system.

The rates presented in this report are based on the diagnosed cases among local residents and must still be considered minimal; that is, less than the

* Almost half of the residents of the city are examined each year by the Mayo Clinic physicians at the clinic, in one of the local hospitals, or at home for emergency house calls. The Mayo Clinic record system, at the time of the study, included the reports for all hospital and other medical diagnostic facilities within the city, with the exception of the State Mental Hospital and the few general practitioners in the community. These physicians in private practice refer practically all patients with disorders of any serious nature to the large staff of specialists at the clinic. Neurologic consultations are routinely arranged for all patients who are examined in other sections of the clinic and suspected of having any serious disease of the nervous system. The clinic's diagnostic file consists of diagnoses made in all Sections and includes surgical and necropsy reports from the Section of Pathologic Anatomy. The diagnoses made by each clinical consultant during the patient's period of registry are cross-indexed by means of a system using IBM punch cards and tabulating devices. It is a relatively simple task to obtain the records of all resident patients for practically any clinical disorder.

"true" prevalence or incidence rates for symptomatic neoplasms in this population. The discrepancy for primary neoplasms is probably small and would be caused by failure of some persons to seek medical care after onset of symptoms or the possibility of misdiagnosis among those who have been examined. The study covers a 10-year period so that time is an adjunct in obtaining a high degree of definitive diagnoses; on the other hand, the resident population is small and the rates for the relatively infrequent disorders included here are subject to appreciable effect from chance fluctuation.

METHOD

The records were reviewed for all patients who were residents* of the city and in whom any primary or metastatic intracranial or intraspinal neoplasm was diagnosed during the 10-year period, 1945 to 1954, inclusive. Data for the statistical analysis, including age, sex, race, type of tumor, date of first Mayo Clinic diagnosis, and course of illness were then abstracted.

The cases were divided into several principal groups and then subdivided by histologic type. Appropriate rates and ratios were computed.

The principal groups included metastatic neoplasms to the brain and spinal cord, primary neoplasms affecting the spinal cord, pituitary neoplasms and primary neoplasms of the brain. The primary tumors of the brain were classified as "certain" when confirmed histologically and "probable" when the diagnosis was strongly suspected on clinical grounds but not confirmed histologically.

Both incidence and prevalence rates were determined. By incidence rate is meant the number of newly diagnosed cases occurring during a specific period (usually a year) per unit of population (usually per 100,000 population). Only the cases newly diagnosed at the Mayo Clinic during the 10-year period were included here. The average annual incidence rate per 100,000 population for the 10 years was determined as follows:

Average annual incidence rate per 100,000 population equals

$$\frac{\text{no. of resident cases diagnosed at Mayo Clinic during 10-year period}}{\text{10 years} \times \text{population of Rochester, Minnesota (in 1950)}} \times 100{,}000$$

For age-specific rates, the number of cases in a specified age group and the population of that age group were used. The rates were adjusted on the basis of the age distribution of the total population of the United States in 1950 to provide greater comparability with rates obtained from other statistical surveys. Furthermore, an approximation of the number of cases that can be expected in the population of the United States is readily calculated by applying this age-adjusted rate to the population of the United States.

Prevalence rate ordinarily refers to the number of patients living at a

* "Resident" includes only those individuals whose domicile was within the city limits of Rochester, Minnesota, for at least one year during the study period; all persons who were known to have moved to Rochester to facilitate treatment of a condition diagnosed prior to taking up residence were excluded.

specified time per unit of population; however, to simplify comparisons with the cancer surveys by Dorn and others,[2,4,5] their definition of prevalence as "the number of patients living at any time during a specified year per unit of population" was adopted here. In this study, the prevalence rate per 100,000 population was determined for the year 1954.

RESULTS

During the 10-year period, 50 cases of intracranial neoplasm were recorded for the resident population (Table 1). Fifty-four per cent of the patients had primary tumors of the brain, 14 per cent had pituitary tumors, and the rest had metastatic tumors to the brain.

There were 12 cases of intraspinal neoplasm. Eight (67 per cent) were diagnosed as primary tumors and 4 (33 per cent) were metastatic. Two of the latter were also metastatic to the brain.

Prevalence Rates. During the calendar year 1954, 22 of the patients with a diagnosis of intracranial or intraspinal neoplasm were alive. Their number and the age-adjusted rates for each site are shown in Table 2. (None of these happened to be a patient in whom there were both intraspinal and intracranial metastatic lesions.)

If these data can be applied to the other populations of the United States, about 46 persons per 100,000 population have primary intracranial neoplasms and 13 per 100,000 population have primary neoplasms of the spinal cord or roots in any one recent year. On this basis, about 77,000 persons in the United States were affected with primary intracranial tumors and about 22,000 had primary tumors of the spinal cord or roots in 1956. Since all the Rochester rates reported here are based on a small population, they must be interpreted with caution when applied to other populations, such as those of the rest of the United States. Consideration must be given to the effect of chance variation.*

Incidence Rates by Age and Sex. In Table 3, the age-adjusted incidence rates by sex and by type of neoplasm are presented; in Table 4, the rates are shown by age.

Incidence rates are higher for females except in cases of pituitary tumors in this series. According to these rates, each year 9.2 persons per 100,000 population develop a primary intracranial neoplasm and each year 17.3 persons per 100,000 population develop a neoplasm affecting the central nervous system. To look at this another way; if one could apply these rates to other populations in the United States, one might expect the following number of neoplasms to be diagnosed in a city of 1,000,000 population per year:

* If it can be assumed that the United States is randomly represented by the Rochester data, there are 19 chances in 20 (95 per cent confidence interval) that the true prevalence rate for intracranial neoplasms was between 25.0 and 77.6 per 100,000 population and that between 42,000 and 130,000 persons were so affected. Similarly, the true prevalence rate for primary intraspinal tumors is between 3.4 and 33.6 per 100,000 population, so that the number with this disorder probably fell between 6,000 and 56,000 persons.

			95 Per Cent Confidence Interval
Primary tumors of the brain	77	92	68–144
Pituitary neoplasms	15		
Primary neoplasms of the spinal cord	25		12– 53
Intracranial metastasis to central nervous system	50	56	32– 90
Intraspinal metastasis	6		
Total number of cases per year	173		124–221

TABLE 1

Intracranial neoplasms diagnosed at the Mayo Clinic in Rochester, Minnesota, resident population, 1945 to 1954, inclusive

Type of Neoplasm	Number of Patients	Per Cent of Total
Primary brain, certain*	22	44
Primary brain, probable*	5	10
Metastatic lesions to brain	16	32
Pituitary	7	14
Total	50	100

* "Primary brain, certain," refers to histologically confirmed neoplasms; "primary brain, probable," refers to cases for which there is strong clinical or surgical evidence of a primary neoplasm of the brain which has not been histologically confirmed.

TABLE 2

Prevalence and age-adjusted prevalence† rates per 100,000 population for neoplasms affecting the central nervous system, by site, for Rochester, Minnesota, population, 1954*

Type of Neoplasm	Number of Patients	Age-adjusted Rate per 100,000 Population‡	
Primary brain, certain	8	24.7	
Primary brain, probable	4	12.4	46.0
Pituitary	3	8.9	
Intracranial metastasis	3	9.7	
Primary spinal cord	4	12.9	
All types	22	68.6	

* Prevalence obtained by accepting all patients who were residents and alive in Rochester, Minnesota, at some time during 1954.

† Rate adjusted for age on the total population of the United States, 1950.

None of the patients with a diagnosis of intraspinal metastasis was alive during 1954.

‡ Ninety-five per cent confidence limits of the age-adjusted rates were computed.

If the primary tumors of the brain and the pituitary tumors are combined, the prevalence rate for the intracranial neoplasms is 46.0 per 100,000 population. The 95 per cent confidence limits for these intracranial neoplasms are 25.0 and 77.6 per 100,000 population.

A similar confidence interval per 100,000 population for the primary neoplasms of the spinal cord is between 3.4 and 33.6 per 100,000 population, the interval for intracranial metastatic lesions is between 1.9 and 28.8. For all neoplasms affecting the central nervous system, the confidence interval is between 42.2 and 105.4 per 100,000 population. (No adjustment was made for the relatively minor effect of age standardization in the computation of the confidence limits.)

TABLE 3

*Average annual incidence rates per 100,000 population for neoplasms of the central nervous system, resident population, Rochester, Minnesota, 1945 to 1954, inclusive, by sex, age-adjusted**

Type of Neoplasm	Male	Female	Total
Primary brain, certain	4.0	8.0	6.2
Primary brain, probable	1.4	1.5	1.5 | 9.2
Pituitary	2.9	0.5	1.5
Intracranial and intraspinal metastasis	3.7	7.1	5.6
Primary spinal cord	2.1	2.6	2.5
All types	14.1	19.7	17.3

* Rate adjusted for age on the total population of the United States, 1950.

For the United States, it is expected that there are, on this basis, about 15,000 new primary intracranial neoplasms annually and about 29,000 new cases of neoplasm affecting the central nervous system.*

In view of the small number of cases in some of the categories, the age-

* The annual expectations at the 95 per cent confidence level would be between 11,000 and 24,000 new cases of primary intracranial neoplasms and between 21,000 and 37,000 new cases of neoplasms affecting the central nervous system.

TABLE 4

Incidence and average annual incidence rates for newly diagnosed intracranial and intraspinal neoplasms in Rochester, Minnesota, resident population, 1945 to 1954, inclusive, by age

Age Group	1950 Population	Primary Brain Tumor, Certain and Probable		Intraspinal* and Intracranial Metastasis		Primary Intraspinal Neoplasm		Pituitary		Total	
		No.	Rate†	No.	Rate†	No.	Rate†	No.	Rate†	No.	Rate†
0–24	11,391	3	2.6	0	0	1	0.9	1	0.9	5	4.4
25–44	8,899	7	7.9	3	3.4	4	4.5	1	1.1	15	16.9
45–64	6,579	8	12.2	13	19.8	2	3.0	0	0	23	35.0
65 plus	2,942	7	23.8	2	6.8	1	3.4	3	10.2	13	44.2
Total	29,811	25	8.4	18	6.0	8	2.7	5	1.7	56	18.8
Age adjusted rate‡	—		7.7		5.6		2.5		1.5		17.3
95% Confidence interval			4.9–11.6		3.2–9.0		1.2–5.3		0.5–3.9		12.4–22.1

* Two of the 18 patients in this group had intraspinal lesions only—one patient in each of the last two age groups.

† Average annual rate per 100,000 population.

‡ Rate adjusted for age on the total population of the United States, 1950.

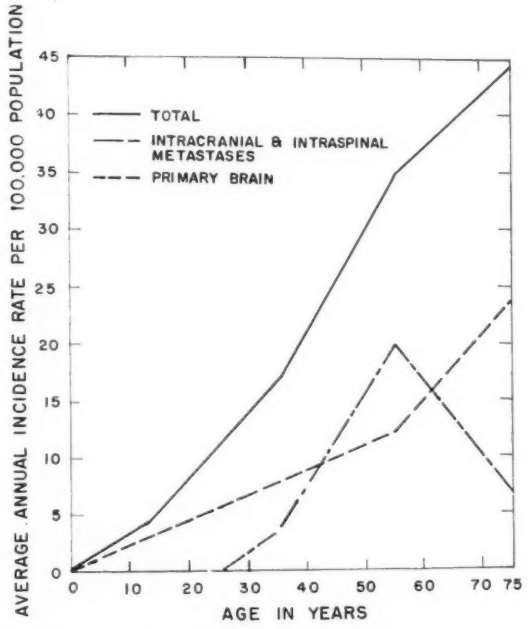

FIG. 1. Newly diagnosed neoplasms of the central nervous system, Rochester, Minnesota, resident population, 1945 to 1954, inclusive, by age.

specific incidence rates are shown for the sexes combined. Fig. 1 illustrates these rates for all the neoplasms, for primary tumors of the brain ("certain" and "probable" cases) and for the intracranial metastatic lesions.

The rate increases steadily with age for primary neoplasms of the brain and for the total group while that for the metastatic group reaches a peak in the 45- to 64-year age group.

Death Ratio. During the 10-year period, there were approximately 2,100 deaths from all causes among the residents of this city. In this same period, there were 37 deaths from intracranial and intraspinal neoplasms; in Table 5,

TABLE 5

Death ratio for intracranial and intraspinal neoplasms, Rochester, Minnesota, resident population, 1945 to 1954, inclusive

Cause	Number	Ratio per 1,000 deaths
Primary intracranial neoplasms	16	7.6
Pituitary neoplasms	3	1.4
Intracranial and intraspinal metastatic lesions	17	8.1
Primary spinal neoplasms	1	0.5
Total	37	17.6

the number and proportion of all deaths from these causes are listed. About 1 per cent of all deaths were caused by primary intracranial neoplasms (brain, meningeal and pituitary) and about 1.8 per cent of all deaths were caused by primary and metastatic intracranial and intraspinal neoplasms combined.

Type of Tumors. (1) *Primary Intracranial.* There was histologic confirmation in 22 of the 27 diagnosed cases. Eight were astrocytomas (1, grade 2; others, grade 3 or 4); 8 were meningiomas; 2 were acoustic neurofibromas; and there was 1 each of parasellar chordoma, myxofibrosarcoma, hemangioma, and retinoblastoma with cerebral extension.

(2) *Pituitary.* There were 7 cases (5 M, 2 F). One elderly male with acromegaly was found to have an acidophilic adenoma at necropsy 12 years after symptoms were first noted. In all the other cases, the diagnosis was chromophobe adenoma.

(3) *Primary Intraspinal.* In 7 of the 8 cases the diagnosis was confirmed histologically; in the other case a diagnosis of a lesion of the cauda equina was based on clinical and roentgenologic evidence alone. Of the 7 tumors confirmed histologically, 3 were neurofibromas, 2 were meningiomas, and 2 were ependymomas.

(4) *Metastatic Intracranial and Intraspinal.* There were 16 patients with metastatic intracranial lesions; 2 of these also had intraspinal metastatic lesions. Two other patients had intraspinal metastatic lesions alone. Of the 18 primary neoplasms reported, 6 were of the breast, 6 were of the lung and the remaining 6 were of various other tissues.

COMPARISONS OF INCIDENCE IN ROCHESTER, MINNESOTA, WITH THAT REPORTED IN OTHER SURVEYS

The incidence of intracranial and intraspinal neoplasms in Rochester, Minnesota, is compared with that of cancer surveys in Connecticut, Iowa, and 10 metropolitan areas in the United States.[2,4,5] These collectively will be referred to as the "other areas." The studies in Iowa and the 10 cities were essentially a measure of the cases of cancer diagnosed or observed during a 1-year period. The Connecticut survey covered the period 1935 to 1951, but is probably most complete for primary tumors of the brain for the period 1947 to 1951. Case reports were obtained from death certificates and all medical sources reporting under a voluntary reporting procedure.

In addition to differences in the methods of collection of cases and the periods covered in the surveys, there are also differences in methods of classification of neoplasms. In Table 6, an attempt has been made to present comparisons for various primary sites in the nervous system. "Primary brain" is probably the only one that is reasonably comparable in the different studies. The very low incidence of pituitary tumors in the Connecticut report suggests that some may have been included with the primary neoplasms of the brain. The rates for "other nervous system neoplasms" in Connecticut (including spinal cord, sympathetic and peripheral nerves),

TABLE 6

Incidence and age-adjusted incidence rates per 100,000 population for intracranial and intraspinal neoplasms for various communities and states, by sex and type

Area	Primary Brain			Primary Spinal Cord			Pituitary Total	Other Nervous System Total
	Total	Male	Female	Total	Male	Female		
A) Number								
Roch. (Minn.) 1945–54	27	7	20	8	3	5	5	
Iowa, rural 1950	45	29	16					5
Iowa, urban 1950	66	46	20					8
Ten cities, 1947–49, white	637	387	250					152
nonwhite	59	29	30					20
Connecticut, 1947–51	382	213	169					97*
Connecticut, 1941–46							6	
B) Rate per 100,000 population (average annual)								
Roch. (Minn.) 1945–54	7.7	5.4	9.5	2.7	2.3	3.0	1.5	
Iowa, rural 1950	3.3	4.1	2.3					0.3
Iowa, urban 1950	5.1	7.4	3.0					0.6
Ten cities, 1947–49, white	5.1	6.3	3.9					1.2
nonwhite	3.0	3.0	2.9					1.0
Connecticut, 1947–51	3.6	4.1	3.0					1.0
Connecticut, 1941–46							0.5	

* Specified as other *central* nervous system.

in Iowa and the 10 cities are lower than the rate for primary neoplasm of the spinal cord alone in Rochester.

The rates for primary brain, primary spinal and pituitary tumors for both sexes combined are somewhat higher in Rochester, Minnesota, than in the other areas. This is probably because of the more complete method of collecting cases in Rochester rather than to any geographic difference in the frequency of the neoplasms. Most of the rates for each sex are also presented in Table 6; the differences noted here will be discussed later.

In Fig. 2 there is a comparison of primary tumors for both sexes combined

for Rochester, Minnesota, and the other areas. The age-specific rates for the other areas are similar to one another and all decrease in the age group 65 years and over. In Rochester, Minnesota, on the other hand, the rate is highest in the oldest age group. It is believed that the diagnosis of brain neoplasms in the older age groups is more complete and more accurate in Rochester, Minnesota, than in the other areas. One important factor in this respect is that about two-thirds of deceased residents are examined at necropsy! It was noted that among the instances at which the first diagnosis of neoplasm was made at necropsy, there were a few patients in whom a

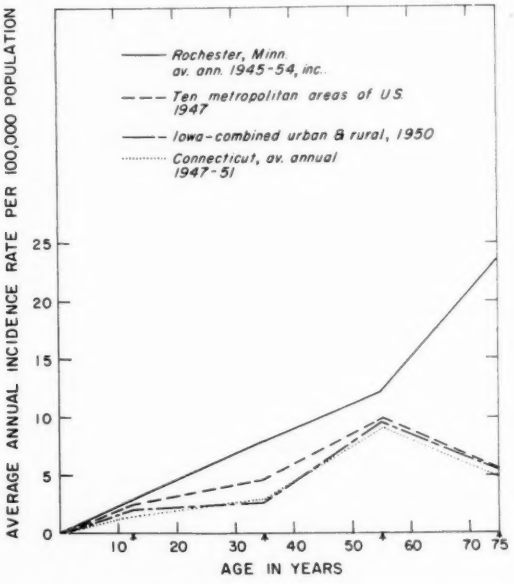

FIG. 2. Primary neoplasms of the brain; incidence rate, by age.

clinical diagnosis of vascular accident was superseded by a necropsy diagnosis of a primary tumor of the brain. The relatively high proportion of the total population in this community in whom a histologic diagnosis is made at operation or at necropsy is believed to account for greater accuracy of diagnosis of intracranial disease and also may account for the increasing incidence rate of primary tumors of the brain with age, contrary to the experience in the other areas and to the experience of others reporting series of surgical cases.

COMMENT

This report is believed to be the most nearly complete population survey of intracranial and intraspinal tumors available to date. Although the 10-year period represented by the study provides 300,000 "person years" of

experience, the resident population is somewhat small to yield relatively stable rates and ratios for uncommon disorders such as those included in this report. The rates by sex, age and type of neoplasm may well be subject to appreciable chance fluctuations. Nevertheless, it is the writer's opinion that the best method of detecting possible associations of geographic, social or racial incidence is to carry out systematic population surveys of which the present study, in conjunction with other recent population surveys[7] might be considered a prototype.

Willis[14] has described the deficiency of the statistics in general hospital series as follows: "The relative frequencies of various tumors in the clinical and necropsy records of a general hospital are not the true relative frequencies, but are influenced by many extraneous factors." Comparisons of surgical series are particularly subject to the hazards of nonuniformity in method and the following examples are presented to illustrate the need for restraint in concluding that differences in such series demonstrate geographic or population influences. In discussing the relative frequency of different types of intracranial tumors, Wilson and Bruce[15] compared Cushing's series with those of Olivecrona in Sweden and several British authors and suggested "they (pituitary tumors) must be far more common in the United States of America than elsewhere," whereas "metastatic growths occur more often (in the British experience) than in the American or Scandinavian series." Cushing's unusual interest and ability in pituitary surgery and his relative, if not absolute, exclusion of cases of metastatic lesions resulted in a series which, first, was not necessarily representative of all brain tumors in the population of the United States and second, should not be compared without qualification to the series of other surgeons.

Earle and associates[3] in their intensive review of brain tumors at the University of Texas Hospital in Galveston, noted a higher proportion of white to Negro patients. Since the ratio was greater than that of the beds allotted to the Negro patients, they suggested that the disproportion might be ascribed to a difference in racial susceptibility for brain neoplasm. There may, indeed, be racial differences as suggested by the higher relative frequency of whites over nonwhites in necropsy series.[10,11] However, this cannot be definitely concluded in the absence of population surveys that are equally thorough for all segments of the population and that take into consideration variations in the age and race distribution and the relative frequency of other causes of death as well.

Primary intracranial tumors accounted for about 1 per cent of all deaths in the Rochester, Minnesota, population, and symptomatic metastatic lesions of the central nervous system were present in almost 1 per cent of all fatal cases. Primary tumors of the spinal cord were generally successfully removed and, therefore, were rarely reported as a cause of death.

The proportion of deaths caused by brain neoplasms in this community was about twice that reported in the national mortality statistics. Although necropsy data may not accurately portray the various causes of death in a

population, it is interesting that brain neoplasms were noted in 1.3 per cent of all necropsies in the series of Newbill and Anderson[10] in New Orleans and in 1 per cent of all necropsies in the extensive collection by Steiner[11] for the Los Angeles County Hospital.

The incidence and prevalence rates for Rochester, Minnesota, were higher for both intracranial and intraspinal neoplasms than those reported in the course of other community surveys for cancer. This is believed to be attributable to the superior medical reporting available in Rochester, Minnesota, rather than to any true difference in frequency in the different communities.

One of the more interesting results of this study is the apparent discrepancy in the age distribution of the primary neoplasms of the brain as compared with that reported from all other sources. Cushing's surgical series as well as those of other investigators usually show the highest incidence about or below age 45 years, and Merritt[9] summarizes most of the clinical reports with "in general, intracranial tumors occur predominantly in early life or in middle life." However, these are clinical series of cases and are not *rates*, that is, they are not related to the population at risk at a given age.

Age-specific incidence rates have been computed in only a few instances. In the surveys in the other communities the rates were found to diminish in the oldest age groups. In the extensive necropsy series reported by Marsh,[8] the highest incidence of gliomas was in the 60- to 70-year age group. In the present study, the rates increased with age and it is suggested that this was caused by the relatively accurate clinical and pathologic diagnoses in the older population in Rochester, Minnesota.

In a few elderly residents of Rochester in whom a clinical diagnosis of cerebral vascular accident was made, a neoplasm was demonstrated at necropsy and the corrected diagnosis was incorporated into the unified record system. This study demonstrates the value of a unified record system and a high community necropsy rate in community surveys of morbidity and mortality.

The observation of an increase in the rate for brain neoplasms with age may have little practical value in so far as the surgical management of the elderly patient is concerned but it may have important implications in so far as future consideration of the pathogenesis of the brain-tumor group is concerned. For most forms of cancer the incidence rate continues to rise beyond age 60 years,[12] and the same has been noted for the primary neoplasms of the brain in the present study.

No special inquiry was made with respect to the family history of patients; routine histories in the charts did not reveal familial cases. Present evidence, including the genetic study of Harvald and Hauge[6] does not disclose any simple hereditary factor responsible for gliomas.

According to Merritt,[9] "the frequency of intracranial tumors is approximately equal in the two sexes, but the statistics of some authors would in-

dicate that the incidence in the male sex is slightly greater than in the female." In the other community surveys, the age-adjusted rates for primary tumors of the brain were higher in males, whereas in the present study the rate is higher in females.

In Steiner's[11] extensive necropsy series at the Los Angeles County Hospital, primary tumors of the brain were noted 1.67 times as often in white females as in white males. In the Charity Hospital necropsy series of Newbill and Anderson[10] the ratios for primary tumors of the brain were twice as high in white females as in white males. In view of the small numbers in the present study, future population surveys will have to be depended upon to clarify the question of any susceptibility for either sex.

Almost all tumors diagnosed as primary neoplasms of the brain in this series were confirmed histologically. Meningiomas comprised a somewhat higher proportion of the total (8 out of 22) than in other reported series, in which they generally account for about 15 per cent of the cases.[16] It is of interest that among the brain tumors observed at necropsy of residents and nonresidents of Rochester, Minnesota, at the Mayo Clinic from 1939 to 1954, 300 (10 per cent of the total) were asymptomatic and among these 300, 100 (33.3 per cent) were meningiomas.[16]

Metastatic tumors comprise about one-third of all the diagnosed tumors of the brain in the present study. This is considerably higher than the 1 to 8 per cent reported in neurosurgical series but is lower than the 62.7 per cent reported in chronic disease hospitals providing terminal care for patients with any type of malignancy.[13] In the present study, it is likely that there is more underreporting of metastatic cerebral cancer than of the primary neoplasms of the brain. The metastatic cases were included only when involvement of the central nervous system was specified on the diagnostic face sheet of the patient's record. In most instances signs of cerebral dysfunction, focal seizures or localized paresis had developed; in several cases, the cerebral metastasis was observed and recorded for the first time at necropsy. It is the author's opinion that if all brain neoplasms in a population were correctly diagnosed and tabulated about 35 to 50 per cent would be metastatic.

SUMMARY AND CONCLUSIONS

1. Incidence and prevalence rates and death ratios were computed for intracranial and intraspinal neoplasms for the resident population of Rochester, Minnesota, for the 10-year period 1945 to 1954, inclusive. The records of the Mayo Clinic were the source of data for the diagnosed cases; these records included the reports of the local hospitals and pathologic laboratories associated with the clinic. It is believed that the survey includes the large majority, if not all, of local residents with diagnosed neoplasms affecting the central nervous system.

2. There were 50 cases of intracranial neoplasm. Twenty-seven of the tumors (54 per cent) were primary neoplasms of the brain and meninges, 7 (14 per cent) were pituitary tumors, and the other 16 (32 per cent) were

metastatic tumors. Twenty-two of the 27 primary tumors of the brain were histologically confirmed: 8 were gliomas, 8 were meningiomas, and the other 6 were of miscellaneous types. Six of the 7 pituitary neoplasms were histologically confirmed: 1 was an acidophilic adenoma and the others were chromophobe adenomas.

3. There were 12 cases of intraspinal neoplasm. Seven of the tumors were histologically confirmed primary neoplasms, 1 was clinically suspected, and 4 were metastatic to the spinal column with signs of cord compression.

4. The prevalence rate based on those patients alive in 1954 was 46 per 100,000 population for primary intracranial neoplasms and 13 per 100,000 population for primary neoplasms affecting the spinal cord. If these rates are applicable to the total population in the United States in 1956, there were about 77,000 persons with symptomatic primary intracranial tumors and 22,000 with symptomatic tumors of the spinal cord or roots in the United States that year. The 95 per cent confidence intervals for these estimates are also presented.

5. Average annual incidence rates (that is, the number of newly diagnosed cases each year) were 9.2 per 100,000 population for primary intracranial neoplasms and 2.5 per 100,000 population for primary neoplasms affecting the spinal cord. On this basis, there should be about 15,000 new primary intracranial neoplasms and about 4,200 new neoplasms of the spinal cord and roots annually in the United States.

6. About 1 per cent of all deaths in the resident population were caused by primary intracranial neoplasms; metastatic tumor to the brain caused about the same percentage of deaths. In only one instance was a primary intraspinal tumor associated with the death of the patient; in the others, the tumor was successfully removed surgically.

7. Incidence and prevalence rates were found to be higher in females for primary neoplasms of the brain and meninges and for metastatic tumors. The rate for pituitary tumors was higher in males, and there was no appreciable difference by sex for primary tumors affecting the spinal cord.

8. The age-specific rates for primary tumors of the brain increased with advancing age; in most other studies reviewed here the rates are said to diminish after 45 years of age.

9. Although the resident population is somewhat small to yield stable rates for subgroups by age, sex and type of tumor, the survey is believed to provide a more accurate picture of the frequency of neoplasms of the central nervous system than heretofore available, because of the high level of the diagnostic services in the community, the unified record system at the Mayo Clinic, and the high proportion of histologically confirmed cases from surgical and necropsy studies.

The author wishes to acknowledge the recommendations and guidance in this study by members of the Sections of Neurology and Biometry, Mayo Clinic, and to

thank Mr. Samuel Marcus and Mr. Irving Goldberg, statisticians, National Institutes of Health, for their assistance and advice in the preparation of the tables.

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EXPERIMENTAL OBSERVATIONS ON THE USE OF RUBBER IN THE TREATMENT OF CRANIOSYNOSTOSIS

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(Received for publication September 26, 1957)

THE surgical treatment of craniosynostosis by the wrapping of inert foreign material around the edges of linear craniectomies parallel to fused cranial suture lines was first reported by Simmons and Peyton[16] in 1947, who used tantalum foil in 2 infants. Prior to this, Lannelongue[12] in France in 1890, and Lane[11] in the United States in 1892, had advocated the use of simple linear craniectomies. However, their method of treatment rapidly became unpopular because of indefinite indications for the operation. In 1927 and again in 1943, Faber and Towne[6,7] re-introduced the use of linear craniectomy in craniosynostosis.

In 1948 Ingraham *et al.*[10] carried out extensive experimental studies on the use of fibrin film, oxidized cellulose gauze (Oxycel), tantalum, methyl methacrylate (Lucite), and polyethylene (Polythene). They studied the tissue reaction to these substances when placed between the margins of artificially created channels in the cranial vaults of dogs and monkeys with survivals from 32 to 315 days. Control linear craniectomies were placed on the opposite side of the cranium in each animal. Bony fusion was found in all control saw cuts examined after 100 days. They concluded that polyethylene, because of its flexibility, transparency, and availability in various sizes of tubing and film, was particularly adaptable for introduction into the gap of artificially made sutures in the cranium in order to inhibit bony healing. They observed that its tolerance by the tissues was equal to that of tantalum or Lucite. They found that fibrin film and Oxycel failed to inhibit bony union across the saw cuts. Tantalum and Lucite were found to be inert and to inhibit bony fusion. However, the weight and opacity of the tantalum to roentgen rays and the rigidity, brittleness and difficulty in molding of the Lucite made the use of these substances difficult.

Following the study by Ingraham *et al.*[9] there were no unfavorable reports regarding the use of polyethylene in the treatment of craniosynostosis until 1956 when Anderson and Johnson[3] noted a reaction to polyethylene film in 2 patients, necessitating its postoperative removal. They were compelled to search for a new method to prevent bony regrowth across a linear craniectomy. They described the use of a thorough application of Zenker's acetic fixative or of a 7 per cent tincture of iodine on the surgically exposed

dura mater in 30 human cases, with favorable preliminary results. It has been the general experience in this clinic that polyethylene material which has been embedded in human tissues has a tendency to become brittle and to crack. Experimental studies by Oppenheimer *et al.*[13,14] and Bering *et al.*[4] have revealed that numerous implanted plastic materials, including pure polyethylene, have induced the development of sarcomas in rats and mice in from 400 to 650 days. These observations noted above have prompted us to consider the use of a soft, pliable, resilient, relatively inert material such as rubber in the treatment of craniosynostosis. This report is concerned with observations on the use of rubber material in chronic animal experiments.

MATERIALS AND METHODS

Fourteen mature cats weighing approximately 2 to 4 kg. were used. Anesthesia was obtained with pentobarbital sodium (U.S.P.) in a solution of 10 per cent alcohol injected either intravenously or intraperitoneally in the approximate dosage of 25 mg. per kg. of body weight. The hair of the scalp was clipped with small animal clippers. The operative procedures were carried out with aseptic technique and were essentially the same as described by Ingraham *et al.*[10] The cranium was exposed through a midline sagittal incision. The pericranium was elevated from the mid frontoparietal areas. Two bi-frontoparietal and bi-parietal burr holes 1 cm. in size were made approximately 2 mm. from the midline. The bi-frontoparietal burr holes usually bisected the coronal sutures and were about 2 to 2.5 cm. anterior to the bi-parietal burr holes. The burr holes were connected unilaterally with Gigli saw cuts, the edges of which were trimmed to right angles, leaving bilateral linear craniectomy defects averaging approximately 2 mm. in width. No bone wax was used in these experiments. Bleeding from the bone was allowed to stop spontaneously.

One linear craniectomy was used as the control and was left untouched following its completion. On the side in which the tested material was used, 2 holes were made with a 1-mm. dental drill, approximately 2 mm. from the lateral edge of the saw cut at each end of the cut. Three types of rubber material were used: 1) latex surgical tubing;* 2) Penrose rubber drains;† and 3) condom rubber.‡

The material to be tested was then wrapped around the lateral edge of the linear craniectomy and held in place with No. 40 stainless steel wire sutures passed through the dental drill holes. The dura mater was not opened. The galea and the skin were closed with interrupted 0000 black silk sutures. There were no operative deaths. One cat in which Penrose rubber had been implanted died 11 days postoperatively with a purulent wound infection. It was autopsied and studied although it was not used in the final consideration of these results.

* Manufactured by Rubber-Latex Products, Inc., Cuyahoga Falls, Ohio, U.S.A. No. 200, size $\frac{1}{8}$ inch I.D., Wall Amber. The manufacturer states that the tubing "contains approximately 95 per cent pure latex rubber, the balance being made up of small quantities of zinc oxide, sulphur, wax and an aliphatic amine."

† Manufactured by Sterling Rubber Co., Ltd., Guelph, Canada. The rubber is a centrifuged, clarified, prevulcanized, natural latex. The manufacturer lists a 2 per cent chemical content made up of the following ingredients: "Sulphur, Accelerator 552, zinc oxide, Antioxident 2246, El-Sixty, soap stabilizer, Butyl Zimate, casein stabilizer, ammonia, and caustic potash".

‡ Manufactured by Julius Schmidt, Inc., New York. The rubber is a natural latex containing only a trace of free sulphur. It was generously supplied by the manufacturer in a powder-free state for use in this experiment.

Pathological Studies. The animals were sacrificed at intervals from 1 to 369 days with pentobarbital sodium (U.S.P.). Immediately after death, the operative areas were inspected and the character of the implanted rubber was noted. The operative area with adjacent brain was then removed *en bloc* by making 2 coronal cuts with an electric autopsy saw, one anterior to the coronal suture, and the other at the level of the lambdoid suture. A transverse cut was then made at the level of the inferior surface of the cerebral hemispheres.

The specimen was placed in a 4 per cent solution of formaldehyde for about 3 weeks. The specimens were then carefully examined under the dissecting microscope. Roentgenographs were taken of the cranial vaults of animals surviving longer than 3 months. Most of the brains, including all specimens with survival periods of 92 days or longer, were then sectioned in coronal slices beneath the operative areas. Adjacent slices were taken for microscopic study. The posterior slices were embedded in paraffin and stained with hematoxylin, phloxine and saffron; Laidlaw's method for connective tissue; Mallory's phosphotungstic acid; and Luxol-fast blue and cresyl violet for myelin sheaths and cellular detail. The anterior slices were placed in Cajal's formol-ammonium-bromide solution for at least 1 week. Sections were cut on the freezing microtome at 20 μ. and were stained for astrocytes by Cajal's gold chloride sublimate method, for oligodendroglia and microglia by Hortega's silver carbonate, and for fat with the Scharlach-R stain.

The rubber material was carefully dissected free from the operative site, and the skulls with attached dura mater were decalcified in nitric acid (20 per cent) and phloroglucinol (1 per cent) and embedded in 8 per cent celloidin. Sections were cut at 35 to 60 μ. and stained with the hematoxylin van Gieson method.

RESULTS (Table 1)

Controls. In 7 cats examined within the first 19 days there was no bony fusion. The 7 cats examined after 92 days all showed solid bony union across the saw cuts. This was demonstrated both grossly and microscopically (Fig. 1), and roentgenologically. The control craniectomy, 369 days after operation, presented gross and microscopic evidence of solid bony fusion. However, the roentgenogram (Fig. 4) revealed only scattered points of bony union.

Rubber Tubing. Latex surgical tubing was used in 2 cats examined at 92 and 369 days (Fig. 2). In both animals the rubber had changed in color from light tan to an opaque tannish-white. The rubber was completely intact and had probably lost a slight degree of its normal resiliency. The tubing was well encapsulated in each case by a tough, white, slightly shiny, fibrous capsule. The underlying dural layers were intimately connected to the connective tissue surrounding the rubber and were moderately thickened. There was a complete prevention of bone formation across the saw cuts. Histological sections in each case revealed the thick fibrous capsule made up of parallel connective-tissue fibers, fibroblasts, and phagocytes. The capsules were intimately merged with the underlying dura mater and the overlying muscle and fascial tissues. The outer layer of the capsules contained a few small blood vessels. New bone was not observed to penetrate the capsules or to have formed from the underlying dura mater or overlying soft tissues.

TABLE 1

Summary of experimental data

Cat	Survival Time in Days	Material Used	Preservation of Rubber	Encapsulation	Dural Thickening	Evidence of Bony Union on Tested Side	Evidence of Bony Union on Control Side	Underlying Brain	Remarks
P55-246	92	latex surgical tubing	good	thick	very slight	none	moderate	very slight gliosis	
P55-245	369	latex surgical tubing	good	thick	very slight	none	moderate	no changes noted	
P55-236	5	Penrose rubber drain	good	very slight	very slight	none	none	no changes noted	
P55-235	11	Penrose rubber drain	—	—	—	—	—	—	purulent operative infection
P55-250	13	Penrose rubber drain	good	very slight	possibly very slight	none	none	no changes noted	
P55-267	19	Penrose rubber drain	good	slight	possibly very slight	none	none	no changes noted	
P55-268	313	Penrose rubber drain	good	moderately thick	very slight	none	marked	no changes noted	
P55-251	368	Penrose rubber drain	good	moderately thick	very slight	none	marked	meningocerebral cicatrix	dura mater and brain wounded at operation
P55-273	1	condom rubber	good	none	none	none	none	no changes noted	
P55-260	2	condom rubber	good	none	none	none	none	no changes noted	
P55-272	7	condom rubber	good	very slight	none	none	none	meningocerebral cicatrix	dura mater and brain wounded at operation
P55-280	183	condom rubber	good	moderately thick	none	none	marked	no changes noted	
P55-281	306	condom rubber	good	moderately thick	very slight	none	marked	meningocerebral cicatrix	dura mater and brain wounded at operation
P55-259	365	condom rubber	good	moderately thick	very slight	none	moderate	no changes noted	

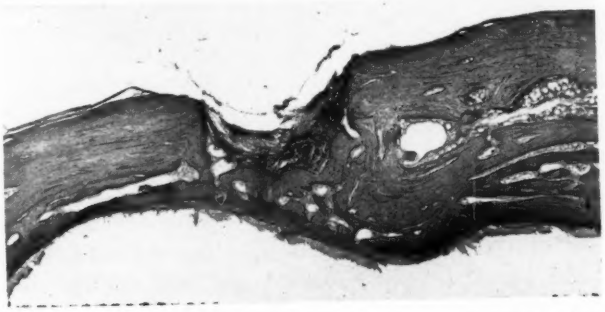


FIG. 1. *Cat P55-246.* Cross section through control linear craniectomy at 92 days. Bone formation is solid across inferior half of the gap. Hematoxylin van Gieson, ×15.

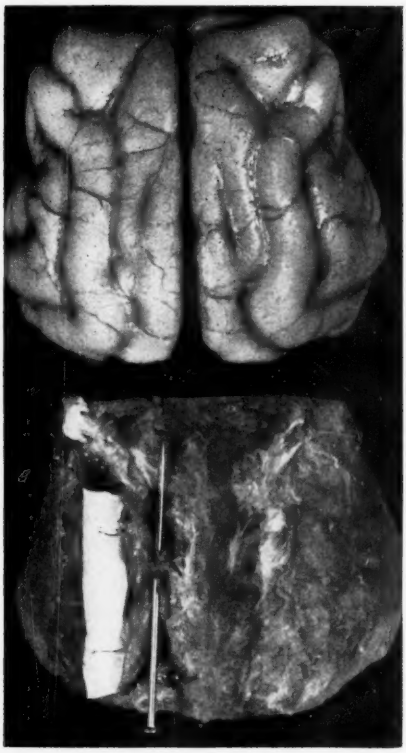


FIG. 2. *Cat P55-245.* Cranium and brain 369 days after implantation of latex surgical tubing around a linear craniectomy on the left and a control linear craniectomy on the right. A pin has been used to reflect the fibrous capsular formation medially from the latex tubing.

The edge of the bone enclosed in the rubber tubing (Fig. 3) presented a moderate underlying cut-in defect. The underlying brain at 92 days had a slight indentation in which region the Cajal gold chloride sublimate stain revealed a slight condensation of astrocytes in the white matter. No significant histological abnormalities were noted in the brain 369 days after operation.

A roentgenogram of the 369-day specimen (Fig. 4) suggested slight bony resorption of the edges of the craniectomy along which the rubber tubing had been placed.

Penrose Rubber. Penrose rubber was placed in 6 cats with survivals of 5, 11, 13, 19, 313, and 368 days. The animal surviving 11 days died with a purulent wound infection and the gross and histological observations are not included in this analysis. The rubber was intact and had changed from a translucent reddish-tan appearance to an opaque yellowish-white appearance in each instance. At 5, 13, and 19 days, a thin layer of loosely organized fibrinous material was noted on the rubber. It was loosely adherent and was readily wiped away.

At 313 and 368 days (Fig. 5) there was a moderately thick grayish-white, slightly shiny, fibrous capsule completely enclosing each piece of rubber. The capsule was intimately attached to the underlying dura mater and to the overlying muscle and fascial tissues. In all animals there was complete prevention of bony union across the saw cuts.

Histological examination at 313 and 368 days revealed a well defined capsular formation of densely packed parallel connective-tissue fibers, fibroblasts, and phagocytes. The capsules were not as thick as those observed around the rubber tubing. There was no evidence of new bone formation invading these fibrous capsules. However, at 313 days there was a finger of bone formation projecting beneath the capsule along the outer layer of the dura mater. After 368 days, the edge of the bone which was enclosed with

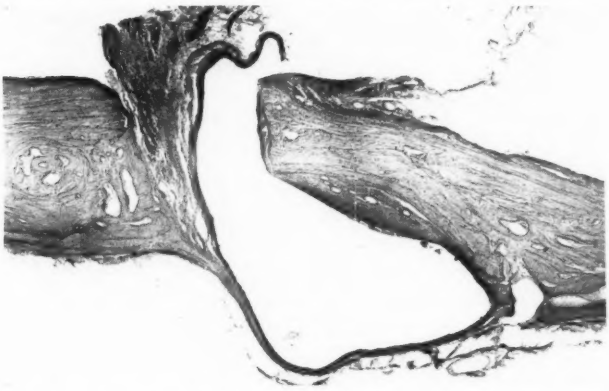


FIG. 3. *Cat P55-246.* Cross section through linear craniectomy in which a section of latex surgical tubing had been implanted 92 days previously. The tubing enclosed the edge of the bone on the right. The fibrous capsule is well developed and there has been no invasion of the capsule by bone formation. Hematoxylin van Gieson, ×15.

Penrose rubber showed a marked cut-in defect, apparently produced by the rubber.

The underlying brains appeared normal in every case except that of the 368-day animal. In that experiment the dura mater had been inadvertently lacerated and there was a healed meningocerebral cicatrix.

Roentgenograms of the 313-day (Fig. 6) and 368-day specimens revealed an absence of bony fusion at any point along the saw cuts around which the rubber had been placed. There was a suggestion of a slight degree of bony resorption of the edge of the craniectomy along which rubber had been placed in the 313-day specimen.

Condom Rubber. Thin, white, translucent condom rubber was placed around the saw cuts in 6 cats with survivals of 1, 2, 7, 183, 306, and 365 days. In each experiment the rubber was found to be intact. It had changed color and was an opaque, faintly yellowish-white. The rubber appeared to have lost a slight degree of its normal resiliency. There were no wound infections in this group. At 1, 2 and 7 days, a thin grayish-white fibrinous material was noted on the surface of the rubber. It was loosely adherent and readily wiped away.

At 138, 306 and 365 days (Fig. 7), tough, grayish-white, slightly shiny fibrous capsule was completely enclosing each piece of rubber. The capsules were judged to be of similar

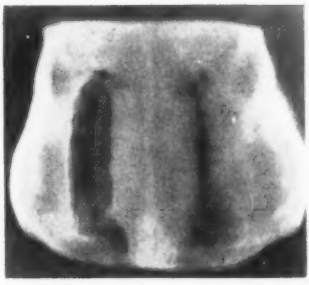


FIG. 4. *Cat P55-245.* Roentgenogram of cranial cap 369 days after implantation of latex surgical tubing in linear craniectomy on left side. The picture is suggestive of bony resorption along the craniectomy. The control craniectomy on the right shows a few points of bony fusion.

FIG. 5. *Cat P55-251.* Cranium 368 days after implantation of Penrose rubber drain in linear craniectomy on the left. Note the fibrous capsule. The control craniectomy on the right is barely visible.

thickness as those enclosing the Penrose rubber. The capsules were intimately attached to the underlying dura mater and to the overlying soft tissues. It was noted that there was a very slight thickening of the underlying dura mater in the 306- and 365-day animals. In each experiment there was complete prevention of bony union across the saw cuts.

Histological examination revealed a fibrous capsule similar to that enclosing the Penrose rubber. At 183 days the capsule appeared definitely thinner than the capsules enclosing the latex surgical tubing at 92 and 369 days and enclosing Penrose drains at 313 and 368 days. The histological preparations noted after 306 and 365 days, in which condom rubber had been embedded, did not present a clear-cut distinction between the fibrous capsule and the adjacent soft tissues. There was no invasion of the fibrous capsules by bone formation. However, small fingers of bone were projecting beneath the capsules along the outer layer of the dura mater (Fig. 8).

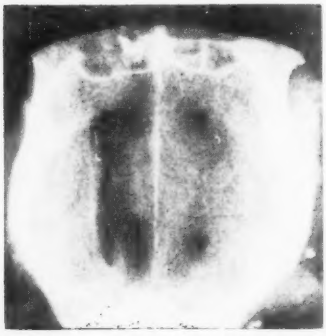


FIG. 6. *Cat P55-268.* Roentgenogram of cranial cap at 313 days in which Penrose rubber had been implanted in a linear craniectomy on the left. Note suggestive bony resorption. The control craniectomy on the right shows good bony fusion.

The underlying brain was normal except for friable meningocerebral adhesions noted at 7 days and at 306 days in instances in which the dura mater and underlying brain had been inadvertently lacerated.

Roentgenograms at 183 (Fig. 9), 306

(Fig. 10), and 365 days (Fig. 11), revealed no evidence of bony fusion across the saw cuts around which the rubber had been placed. In the 306- and 365-day specimens there was a suggestion of a slight degree of bony resorption of the edges of the craniectomy along which the rubber had been placed.

FIG. 7. *Cat P55-259.* Cranium and brain at 365 days in which condom rubber had been implanted in a linear craniectomy on the left. There is no evidence of bony fusion across the gap protected by the rubber. The control craniectomy on the right showed solid bony fusion.

DISCUSSION

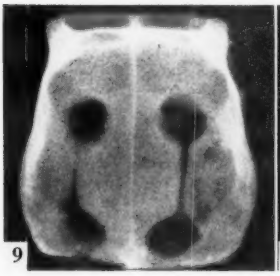
The gross inspection and radiological examinations of these chronic animal preparations revealed that the rubber material tested was effective in the prevention of bony union in linear craniectomies in every case up to 369 days. Excluding one operative infection, there were no untoward gross reactions to the rubber. Histological examinations did reveal a slight foreign-body reaction and/or capsular formation around the rubber in every specimen. This reaction was not considered severe enough in any case to contraindicate the use of rubber material. The presence of the shiny fibrous capsule in every animal with a survival of longer than 19 days is in agreement with the work of Cone *et al.*[5] and of Pudenz and Odom[15] on dural substitutes. They found that a capsule formed around every type of material used as a dural prosthesis, no matter what material. It was interesting to note the degree of variability of bony fusion across the control craniectomies, as revealed roentgenographically. The cranial vault of the animal surviving 183 days (Fig. 3) had a greater degree of bony fusion than the animal surviving 369 days (Fig. 4).

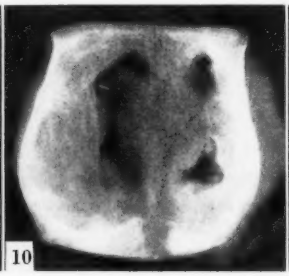
The suggestive bony resorption in the linear craniectomies wrapped with rubber as noted in the roentgenograms of several of the animals with survivals longer than 300 days is a noteworthy observation. It may indicate that the rubber not only prevents bony union but that it also produces in

FIG. 8. *Cat P55-259.* Cross section through linear craniectomy at 365 days in which condom rubber had been interposed. The rubber enclosed the edge of the bone on the right. There is a well formed fibrous capsule without evidence of bony fusion. However, small fingers of bone are seen projecting beneath the capsule along the surface of the dura mater on the right. Hematoxylin van Gieson, ×15.

some unknown manner a slight degree of bony erosion. This characteristic may possibly be considered as a desirable factor in favor of the use of rubber in the treatment of craniosynostosis. The absence of underlying brain damage (excluding the inadvertent operative woundings) indicates that rubber is a safe material for embedding in tissues in close proximity to cerebral tissue.

Abbe[1,2] in 1895 and in 1897 reported 2 cases in which he placed a sheet of rubber on the human brain in an attempt to prevent the reformation of meningocerebral cicatrices. He noted no untoward reactions, even with a 3-year follow-up on one of the cases. The only known experimental study of rubber material applied to the surface of the brain was carried out by Giangrasso[8] in 1940. He implanted sheets of rubber material as dural prostheses in 6 dogs, with survivals from 15 days to 2 years. He noted no un-





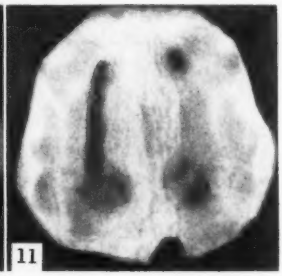


FIG. 9. *Cat P55-280.* Roentgenogram of cranial vault at 183 days in which condom rubber had been interposed in a linear craniectomy on the right. It has completely prevented bony fusion across the gap. The control craniectomy on the left shows several points of bony fusion.

FIG. 10. *Cat P55-281.* Roentgenogram of cranial cap at 306 days in which condom rubber had been interposed in a linear craniectomy on the left. Note the strongly suggestive bony resorption. The control craniectomy on the right shows bony fusion.

FIG. 11. *Cat P55-259.* Roentgenogram of cranial cap at 365 days in which condom rubber had been interposed in a linear craniectomy on the left side. Note absence of bony union. The control craniectomy on the right side shows obvious bony fusion.

toward effects of the rubber. Neither Abbe[1,2] nor Giangrasso[8] specified the type of rubber that they used.

Three types of rubber were used in the present experiment: latex surgical tubing, Penrose rubber drains, and condom rubber. As previously noted the Penrose rubber is free from lead but contains a 2 per cent chemical content. The Schmidt condom is similarly a latex rubber but prepared by a method that gives a film of pure rubber with only a trace of sulphur. The latex surgical tubing is 95 per cent latex rubber. Rubber may be sterilized either by boiling or by autoclaving and thus has an advantage over such materials as polyethylene which cannot be autoclaved.

In a review of the literature I have found no report of a clinical or experimental case in which embedded rubber has produced a true neoplastic growth.

In view of the one operative infection and the usual slight foreign-body reaction to the rubber, it is suggested that clinical application of this experimental study be carried out with the utmost care in asepsis and surgical technique, and that local antibiotics should be instilled in the operative wound. A word should be said regarding the technique of interposing the rubber between the linear craniectomies. The latex surgical tubing, when split longititudinally, molded very readily around the edge of the bony wall. Unfortunately, it is probably too thick for clinical use. However, no untoward behavioral or pathological reactions were noted from its use in this experiment. The condom rubber was found to be very easily manipulated and was readily wrapped around the bony edges and held in place with fine No. 40 stainless steel wire sutures passed through small 1-mm. dental drill holes. The combined qualities of thinness, durability, minimal foreign-body reaction, and ease of use of the condom rubber have led us to consider that it is probably the most satisfactory material to recommend for clinical use. The absence of the 2 per cent chemical content which is present in the Penrose rubber is in favor of the use of the condom rubber over the Penrose rubber.

SUMMARY AND CONCLUSIONS

1. Experimental observations were made on the use of latex surgical tubing, Penrose rubber drains, and condom rubber interposed in linear craniectomies in 14 mature cats with survival times from 1 to 369 days.

2. The rubber material tested was effective in preventing bony union across the linear craniectomies in every case.

3. Gross and histological studies revealed that rubber is well tolerated by the tissues of the cat. A slight insignificant foreign-body reaction and/or capsular formation was noted in every case. (There was 1 animal with an operative infection which was excluded in the final analysis of these data.)

4. Control linear craniectomies in the 7 cats with survivals of 92 days or longer all showed bony fusion.

5. This study indicates that condom rubber would be a satisfactory and desirable material for interposition in artificial cranial sutures in the treatment of clinical cases of craniosynostosis.

I wish to thank Dr. W. V. Cone for his criticisms and suggestions in the preparation of this study.

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CIRCULATORY CHANGES FOLLOWING OCCLUSION OF THE MIDDLE CEREBRAL ARTERY AND THEIR RELATION TO FUNCTION*

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(Received for publication September 26, 1957)

THE sudden onset and rapid recovery from a neurological deficit is relatively common in the course of cerebrovascular disease in man. Thereafter recurrent attacks of a similar nature may occur with a varying degree of recovery.[1,3,4,7,26] Careful review of the history together with observation at the bedside show that a number of factors may precipitate these transient ischemic attacks. Commonly such attacks are preceded by impaired cardiac output or a fall in blood pressure.[24] More rarely anoxia appears to have provoked an attack. A well-defined understanding of these hemodynamic crises is important in planning rational treatment but the sequence of events in the cerebral circulation during these attacks is not yet fully understood.[25]

Pathological studies have clarified the anatomical background but have not resolved the physiological basis for these transient ischemic attacks.[7,15] In general, a patchy softening is present in the distribution of the diseased vessel which usually is found to be the internal carotid or basilar artery and, less commonly, a vessel comparable in size to the middle cerebral artery. Occasionally cerebral infarction is found without demonstrable occlusion of a cerebral vessel. The functional deficit present in life is frequently far greater than can be accounted for by the anatomical extent of the lesion and only hypothesis can be offered to explain recovery from preceding attacks. Anatomical studies have shown the rich supply of small arterial anastomoses which supply additional adjustments in the cerebral collateral circulation other than the circle of Willis.[30] Presumably these vessels play a major role in supplying the circulatory demands of the compromised territory.

In order to define the nature of the neurological deficit following vascular occlusion a series of acute experiments have been made while recording

* These studies were aided by a contract between the Office of Naval Research, Department of the Navy and Harvard University, NR 115–401 and grants from the National Institute of Neurological Diseases and Blindness B-1044, 1956–1957 and from the Milton Fund, Harvard University, 1956–1957.

Opinions expressed are those of the author and do not necessarily represent those of the Department of the Navy.

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With the technical assistance of Robert C. Struzziero.

localized changes in blood flow and oxygen tension from several areas of the brain during occlusion of cerebral vessels.[6,16,19–21] Following occlusion of a cerebral vessel there is reduction of the cerebral oxygen tension within the territory of its supply, the severity of which depends on the effectiveness of the collateral circulation provided by arterial anastomoses. These arterial anastomoses may be observed in the pia with a microscope.[11,20] For example, when the middle cerebral artery is occluded in the monkey the collateral circulation is provided largely from arterial anastomoses in the pia derived from the anterior and posterior cerebral arteries. These vessels measure 50–250 mμ. in size. The most important stimulus for increase in the collateral circulation appears to be a localized reduction in intraluminal pressure so that blood flows from high- to low-pressure areas; local reduction in pH and oxygen tension are less potent stimuli.[20]

Cortical ischemia can proceed to the point of failure of the electroencephalogram and the production of a potential of injury caused by depolarization of nerve cells without damage to vessels, but prolongation of ischemia beyond that point causes anoxic damage to the vascular endothelium, resulting in stasis beginning in the venules. Soon after its production this stasis is reversible by such measures as increasing the blood pressure and the administration of heparin. Infarction appears to result primarily from damage to vascular endothelium with resulting edema, hemoconcentration, sludging and stasis.[6]

Experiments in monkey and later in man[22,23] demonstrate the high consumption of oxygen and small reservoir of oxygen in healthy cerebral tissue. Polarograms in monkeys[5,20] and in man[23] show that oxygen tension and oxygen metabolism are reduced in freshly infarcted cortex but in bordering zones the oxygen tension is the same or higher than in undamaged regions of the brain. In man, under local anesthesia measurements of cortical oxygen tension were correlated with the functional state of the cerebrum as judged by clinical tests and electroencephalogram as the carotid artery was occluded in the neck. Reduction of the cortical oxygen tension to low levels results in temporary impairment of cortical function and slow activity in the electroencephalogram. If the collateral circulation is adequate, compensation begins within 45 seconds of occlusion and the oxygen tension rises towards the steady state prior to release of the artery and functional impairment does not occur. After occlusion of a cerebral vessel the collateral blood flow continues to increase for many days. In the monkey, a relatively controlled degree of cerebral infarction in the Sylvian region may be produced by placing a clip on the parasellar portion of the middle cerebral artery and lowering the blood pressure to 70 mm. of mercury or lower.[20,27,29]

Occlusion of the middle cerebral artery for periods of less than 15 minutes produces only temporary impairment of motor function. To produce a degree of impairment as severe as that associated with permanent occlusion it is necessary to occlude the vessel for at least 50 minutes;[13,14] in this study,

however, a few animals with permanent occlusion of the middle cerebral artery had transient hemiplegia without severe infarction.

The scope of the present paper is to report direct microscopic observations of pial blood flow and oxygen tension in the unanesthetized chronic preparation following occlusion of the middle cerebral artery for as long as 9 weeks and to correlate these observations with the recovery from any functional disorder. In addition, attempts were made to provoke transient ischemic attacks in the convalescent phase by anemically and chemically induced hypotension and by anoxia and to observe any modification of these events by anticoagulant drugs.

METHODS

Twenty-three monkeys (*Macacus mulatta* and *Rhesus cynomologus*) were studied in the present series of experiments. The middle cerebral artery was occluded in 11 experiments without the use of anticoagulant drugs; in an additional 12 experiments anticoagulant drugs (heparin or Dicumarol) were administered prior to the time of occlusion and were continued throughout the period of observation. Heparin was administered by intramuscular injection in doses of 200–500 U.S.P. units per kg. of body weight at 12-hour intervals. The clotting time was consistently found to range between 40 and 90 minutes. Dicumarol (3,3-methylenebis-4-hydroxycoumarin) was administered daily by gastric tube in doses of 0.3–3 mg. per kg.; with this dosage the prothrombin time varied between 40 and 80 seconds, compared to control readings of 13 seconds.

The animals were anesthetized with Nembutal, 0.5 mg. per kg. of body weight, and in some instances chlorpromazine, 2 mg. per kg., was also given to aid in lowering the blood pressure. The head was held firmly in a McGill head holder after shaving the scalp. A hemispherical skin flap was turned down over the zygoma followed by a temporal muscle and periosteal flap using aseptic technique (Fig. 1D).

A trephine hole was made overlying the frontal or parietal operculum of the Sylvian fissure. The trephine hole was tapped and a tight-fitting window,[8] measuring 15 mm. in diameter, was screwed into the skull (Figs. 1D and 2C). The window in the skull was machined according to my specifications by the Harvard workshop and was modified after that described by Sohler *et al.*[28] in 1941.* The glass window was recessed in its brass frame so that the window-glass could be irrigated externally with saline at 98°F. in order to prevent heating of the brain by the source of light (Fig. 2C). Photographs and observations were made through a pool of saline in the glass window as this was found to minimize reflections of light. Two water-tight screws could be removed temporarily from the frame of the window in order to insert polarographic electrodes for making measurements of oxygen and for irrigating the brain.

With the window screwed firmly in place, a large osteoplastic bone flap was fashioned with the window remaining in its center. The bone flap was made by means of a small trephine and a Gigli saw. The dura mater was removed widely and the endosteum of the bone flap was scraped away to prevent growth of fibrous tissue

* Dr. Herbert Jasper of the Montreal Neurological Institute kindly loaned us a window of his own design for use with cortical electrodes. This model was also helpful in designing the window used in these studies.

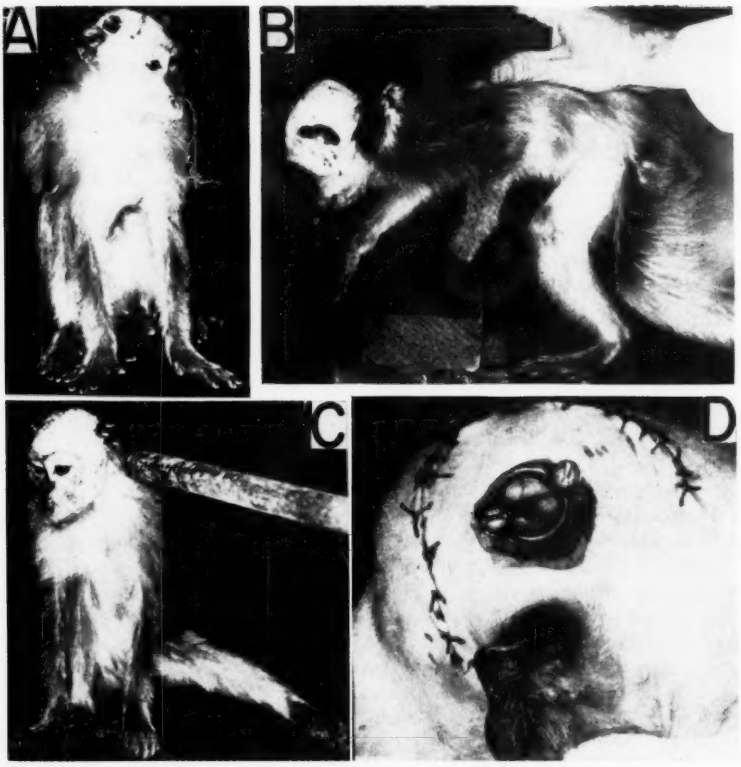

FIG. 1. (A) To show rapid recovery of an animal treated with heparin 24 hrs. after occlusion of middle cerebral artery. (B) The hopping and placing reactions are absent in the left forelimb. (C) A left homonymous hemianopia is present. (D) Close-up view of same animal to show window in place and scalp incision used to permit occlusion of middle cerebral artery. The lateral portion of brain beneath window shows perivenous hemorrhages.

at the edge of the window. The Sylvian fissure was exposed and the frontal lobe was elevated gently with a small malleable retractor. A spot-light was focused on the middle cerebral artery and it was occluded by two or three silver clips. Lowering the blood pressure at the time of surgery was accomplished by the administration of chlorpromazine in doses of 2–5 mg. by lowering the feet on a tilt-table and by the removal of blood. In 3 experiments the vessel was sectioned after the clips had been put in place, in 2 experiments four or five anastomotic branches were occluded on the cortex and in 2 experiments the anterior cerebral artery was occluded in addition to the middle cerebral artery. The tip of the temporal lobe was removed by subpial resection in order to provide internal decompression.

The bone-flap with the window in the center was wired into place so that the window overlay the ischemic cortex in the distribution of the middle cerebral artery. The periosteum, the temporal muscle and the scalp were then closed in layers with

interrupted sutures and the window was exposed through a tightly fitting circular incision made in the scalp. The brain under the window could be irrigated with warm saline. Penicillin, 300,000 units, and streptomycin, 0.2 mg., were given by intramuscular injection after surgery. With practice the whole procedure was completed in 3 hours.

The systolic blood pressure was estimated approximately with a sphygmomanometer and a narrow cuff placed around the lower abdomen with a pad compressing the abdominal aorta. The systolic pressure was taken to be the point at which the femoral pulse was obliterated. This method of sampling the systolic blood pressure obviated puncture of the femoral artery.

Measurements of cortical oxygen tension were made by the polarographic method, using sterile electrodes described in previous communications.[22,23] The anode (silver-silver chloride) was insulated with Teflon except for its last 1 cm. which was freshly chlorided prior to use. The anode was inserted into the muscle of the scalp through the skin flap. The cathodes, or active electrodes, consisted of finely tapered and exposed tips of 0.01-inch platinum wires insulated with Teflon. They were inserted into the cortex through screw holes built into the skull window for that purpose. The exposed portion of wire (1–2 mm. in length) was carefully scraped clean with a scalpel before use but care was taken so that the portion of exposed wire remained unchanged. In this way calibrated readings in electrical units could be compared at different times. It has been found that electrodes insulated with Teflon give readings that are uniform from day to day providing the surface

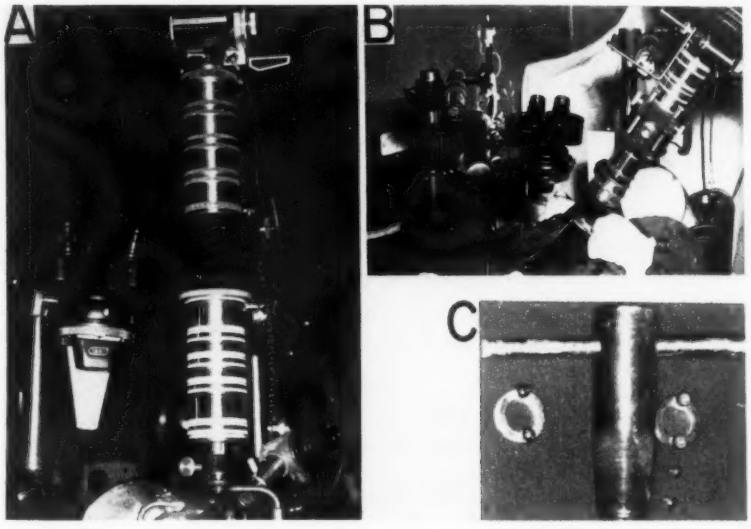

FIG. 2. (A) To show stereoscopic microscope and camera arranged for high-power macrophotography. With this arrangement the image is magnified 180×. (B) To show camera in use at operation. The window is in place within the skull below the drapes. The polarograph and the stereoscopic microscope are to the left of the picture. The electronic flash attachment is to the right behind the camera. (C) To show trephine, tap and modified Forbes window. The glass is recessed so that the window may be cooled externally. The screws have been removed from the window on the right to show how electrodes may be inserted and the brain beneath the window irrigated.

area of the exposed wire is not altered and the metal is kept scrupulously clean.

The principle of the polarographic method requires a potential of 0.6 volt supplied to the anode by a potentiometer. The platinum electrode was connected to the positive pole of the potentiometer through a resistance of 1.2 million ohms. On either side of this resistance leads were taken to the input of an Offner 8-channel Type D3 polygraph. The output from the polarograph was wired directly to the Type 9138 Input Coupler of the inkwriter after calibration with a microammeter.[19,22,23] The electroencephalogram was recorded from the scalp by bipolar needle electrodes placed in both frontal and occipital positions.

Observations of the pial circulation were made with either a Spencer stereoscopic microscope having various magnifications from 18 to 144 times (Fig. 2A) or a monocular Bausch and Lomb microscope permitting magnification up to 480 times. Either microscope was mounted on an adjustable arm. When high-power observations were made a Nicholas illuminator with a blue filter and condenser was used. The illuminator was mounted on an adjustable hinged arm and the light beam was focused on the cortex. In order to avoid heating of the brain the window was irrigated externally with saline.

Macrophotographs (no ocular lens used) were taken with a Praktina camera (VEB Kamera—Werke Niedersedlitz, Dresden, Edgar-André Strasse 56) modified for this purpose.* The camera was mounted on an adjustable hinged arm in such a way that an extension bellows was used for fine adjustment of the focus in a manner similar to that used with a microscope (Fig. 2A, B). Extension tubes were added so that the length of the tubes could be adjusted from 30 to 45 cm. Four lenses were used, each fitted with an adjustable diaphragm. The focal length of the lenses was 50 mm., 25 mm. and 15 mm., and in addition a Bausch and Lomb 10× objective was used. With this arrangement direct magnification from 18× to 180× could be obtained on the ground-glass focusing screen. Anscochrome 35 mm. daylight film was used in the camera and the color transparencies were enlarged on a $3\frac{1}{4}'' \times 4\frac{1}{4}''$ contrast process Panchromatic intermediate negative. Positive prints at various enlargements were made from this. By this means a macrophotograph 180× could be printed satisfactorily on a $5 \times 7''$ print with a resulting magnification of 720×. All macrophotographs were taken with an electronic, high-speed strobe unit having a flash duration of 1/1000 of a second and producing a daylight color flash of 5,600 K° temperature.† The strobe unit was focused on the window from a distance of 10 cm. and provided intense illumination of such short duration that the brain was not heated. The camera was fitted with a reflex view-finder and ground-glass focusing screen adjusted to eye-level so that the photographic field could be viewed as the exposure was made. The animals were examined daily and notes were made of their degree of alertness, respiratory rate, size and reaction of the pupils, visual fields and tendon jerks together with their motor and sensory performance (Fig. 1A, B, C).

With these methods daily observations and photographs of the pial circulation were correlated with the electroencephalogram, cortical oxygen tension and any functional deficit in the restrained but unanesthetized animal. In one animal 8 ml. of a saturated solution of trypan blue was injected intravenously 48 hours after occlusion in order to estimate any increased vascular permeability. The brains were

* Mr. Leo Goodman, Medical Photographer, The Mallory Institute of Pathology, Boston City Hospital, assisted in modifying this camera for high-power macrophotographs.

† Dejur-Ansco Corp., 45-01 Northern Blvd., Long Island City 1, N.Y.

removed immediately after death or sacrifice, photographed, fixed in 10 per cent formalin and embedded and stained for microscopic study. Details of the nature of changes in local hemodynamics from day to day as well as histological studies of the brain will be presented in subsequent papers.[17,18]

RESULTS

General Observations. In the experiments reported here occlusion of the middle cerebral artery near the circle of Willis produced regularly a functional deficit which varied in severity from a transient (4–5 days) faciobrachial monoparesis with loss of hopping and placing reactions and depressed tendon jerks to severe flaccid hemiplegia, with hemianopia and hemianesthesia. The forelimb was consistently weaker than the hindlimb. In those animals with a severe defect there was steady functional improvement after the first week, at which time the hemiparetic limbs became spastic and the reflexes increased. Some mild weakness and absence of hopping and placing reactions in the forelimb persisted for 9 weeks. In some experiments the hemiplegia progressed to stupor and death within the first 72 hours because of brain swelling with herniation and brain-stem compression. In 2 animals in whom brain swelling was severe and whose respirations were slowed the inhalation of 100 per cent oxygen by nasal catheter appeared to reduce brain swelling and the animals survived. In general, the group of animals with severe functional defect were those in whom the systolic blood pressure fell to 70 mm. of mercury or lower. Reduction of the systolic blood pressure below 60–70 mm. of mercury at the time of occlusion produced regularly infarction of the pre- and postcentral cortex adjacent to the Sylvian fissure visible through the skull window. If the blood pressure was maintained above 90 mm. of mercury a mild, transient hemiparesis with complete recovery occurred usually but not invariably. In 1 animal in whom severe infarction occurred without a fall in blood pressure there was an anomaly of the anterior cerebral artery which was derived solely from the occluded middle cerebral vessel with consequent reduction of collateral vessels. The severity of the neurological defect could be increased regularly by occlusion of the anterior cerebral or opposite middle cerebral artery in addition to the middle cerebral artery, or by occluding four or five of the major cortical anastomoses at the cortex.

Using magnifications above 100× of either the microscope or the camera it was possible in the unanesthetized animal to observe capillaries in the pia and immediately subjacent cortex and to identify individual red and white blood cells in zones of stagnant flow.

Circulatory Changes Accompanying Transient Hemiplegia Without Infarction. In those animals in which the blood pressure was well maintained and a mild transient hemiparesis was present for 4–5 days followed by complete recovery there was a critical slowing of the pial circulation without significant stasis but with a marked change in the color of the terminal arterioles and veins. The veins became dark blue and the arterioles a brown-

blue instead of their normal orange-red color. The large arteries did not change in color. The capillaries were less prominent and the terminal venules appeared dark at the points where deep perforating cortical branches joined them at the surface (Figs. 3A and 4B, D). The difference in arterial and venous color was increased compared to the normal appearance of the cortex before occlusion of the middle cerebral artery. The normal axial flow of red and white blood cells was altered in venules and there was margination of white cells with a granular appearance of the arterioles. Many of the capillaries were filled with plasma flowing slowly with periodic resumption of flow of red cells through them. The marked cyanosis of the cortex persisted for the first 4–8 hours and was then followed by progressive

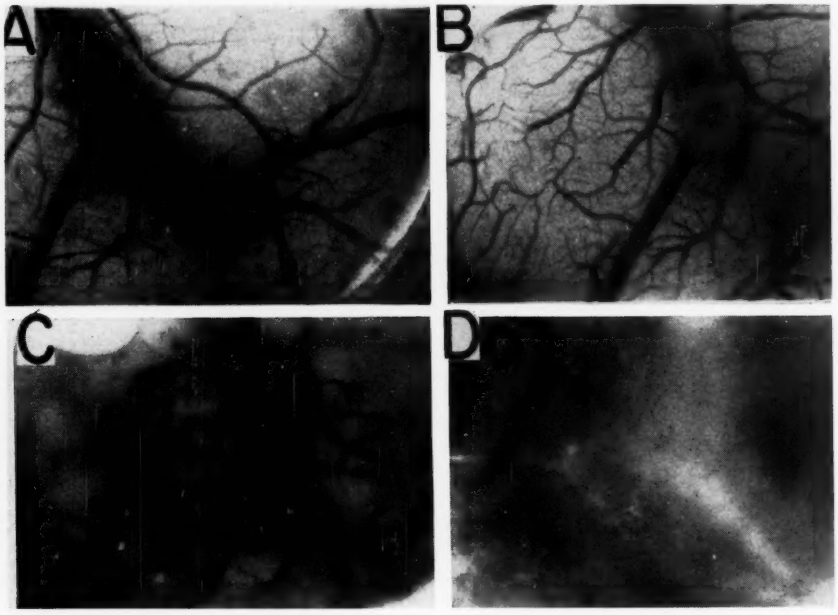

FIG. 3. Serial macrophotographs to show changes in collateral circulation accompanying transient hemiplegia produced by lowering the systolic blood pressure in a monkey recovering from occlusion of right middle cerebral artery.

(A) Macrophotograph (15×) of right pre- and postcentral gyri made 2 hrs. after occlusion. The systolic blood pressure was 110 mm. Hg and collateral circulation was well established. There is some perivascular hemorrhage.

(B) 24 hrs. later hemorrhages have been absorbed and circulation is restored. Mild left hemiparesis was present; systolic blood pressure was 104 mm. Hg. Blood pressure was lowered to 60 mm. Hg for 2 hrs. by administration of hexamethonium, resulting in failure of collateral circulation and development of complete left hemiplegia.

(C) 24 hrs. after administration of hexamethonium (48 hrs. after vascular occlusion) ball-hemorrhages have occurred because of restoration of collateral flow to vessels damaged by ischemic anoxia. Severe left hemiplegia and hemisensory defect were present.

(D) 4 days later (5 days after vascular occlusion) hemorrhagic zone is being invaded by clouds of white blood cells and there is recovery from hemiparesis except for absent hopping and placing reactions.

restoration of blood flow by way of the collateral circulation derived from arterial connections with the anterior and posterior cerebral arteries (Figs. 3B, 4A, and 5A, B, C, D). Hyperemia of the cortex occurred 8–72 hours after occlusion with dilatation of arterioles and venules and rapid flow in the capillary network. Restoration of flow to vessels damaged by ischemic anoxia produced perivascular hemorrhages and diapedesis which usually were absorbed by white cells or washed away by cerebrospinal fluid within

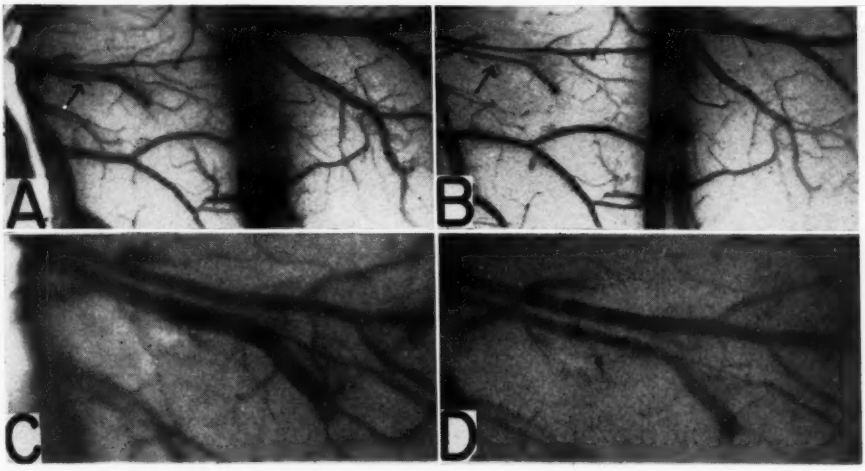

FIG. 4. To show collapse of collateral circulation and production of transient hemiplegia by rapid removal of 15 cc. of blood from femoral vein in an animal that had recovered from occlusion of left middle cerebral artery.

(A) Macrophotograph (16×) of postcentral gyrus made 24 hrs. after occlusion. Blood pressure was 100 mm. systolic and collateral circulation was well established. A mild right hemiparesis was present.

(B) Same field 5 min. after removal of 15 cc. of blood to show narrowing of arterioles and venules. Complete right hemiplegia was present.

(C) High power view (48×) of venule indicated by arrow in A prior to removal of blood.

(D) High power view (48×) of same venule after removal of blood to show collapse of capillary network.

3–5 days (Fig. 3A, B, C, D.) The pia and cortex then appeared normal save for scattered brown pigment within perivascular white cells. During the stage of cyanosis the cortical oxygen tension measured 40–75 per cent of readings prior to occlusion. The oxygen tension returned to normal 8–72 hours later and supernormal levels caused by visible reactive hyperemia were common. The electroencephalogram follows the state of the circulation closely. During the first 8 hours reduction in amplitude and slowing was recorded commonly from the ischemic zone with a rapid return to normal if there was good compensation of the collateral circulation (Fig. 6H). Functional recovery, however, lagged consistently behind restoration of cortical blood flow, oxygen tension and the electroencephalogram for 1 to 4 days.

Circulatory Changes Accompanying Severe Hemiplegia with Infarction.

Severe or prolonged hemiplegia was associated regularly with some degree of infarction visible in the living cortex in spite of eventual functional recovery. In this group of experiments the oxygen tension in the infarcted zone was reduced to 5–20 per cent of normal within the first 24 hours and the cortex presented a pale, cyanotic appearance because of collapse of the

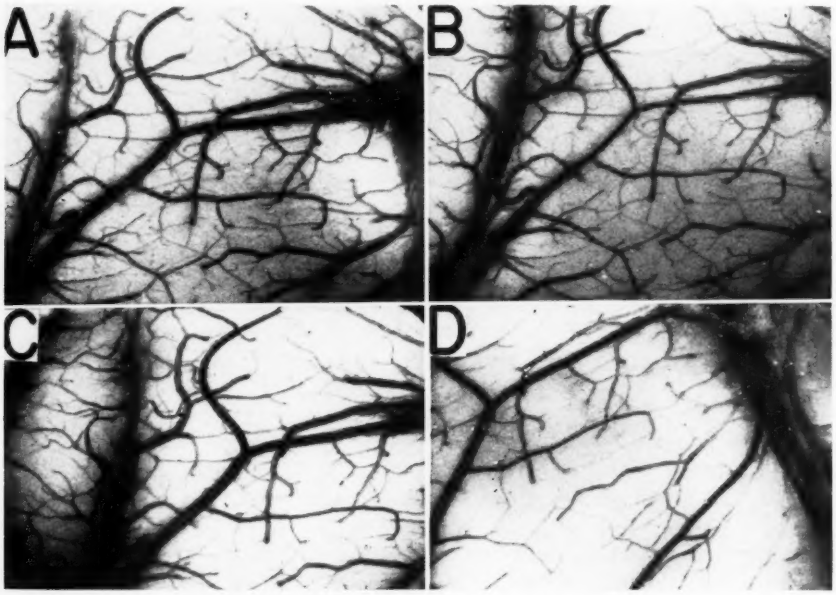

FIG. 5. To show importance of intraluminal pressure in delayed adjustment of collateral circulation.

(A) Macrophotograph (15×) of posterior parietal region made 24 hrs. after occlusion of middle cerebral artery with blood pressure of 100 mm. The right arm of Y-shaped artery in center of the field forms an arterial anastomosis with middle cerebral artery and is widely dilated. The lower arm of Y-shaped vessel is a branch of posterior cerebral artery.

(B) Same vessel 2 min. after rise in blood pressure was produced by administration of 0.5 cc. of epinephrine intravenously. The sudden increase of intraluminal pressure resulted in marked constriction of arterial anastomosis to the right (Bayliss effect).

(C) 10 min. later rise in blood pressure and constriction of vessel have subsided.

(D) Same field 10 min. after occlusion of vertebral arteries. There is collapse of the vascular network and stasis is beginning in lower right corner.

vascular network. Severe ischemic anoxia of this degree was accompanied regularly by brain swelling, and clumping, segmentation and stasis of red cells in venules which occasionally progressed within the next 4 days to involve small arteries (Figs. 7 and 8). White thrombi caused by clumping of platelets were seen but their appearance changed commonly with local alterations of flow (Fig. 7C, D). Arteriolar stasis resulted commonly in progressive brain ischemia and swelling, occasionally producing a vicious cycle which terminated in death from brain edema (Fig. 9A, B). Rarely

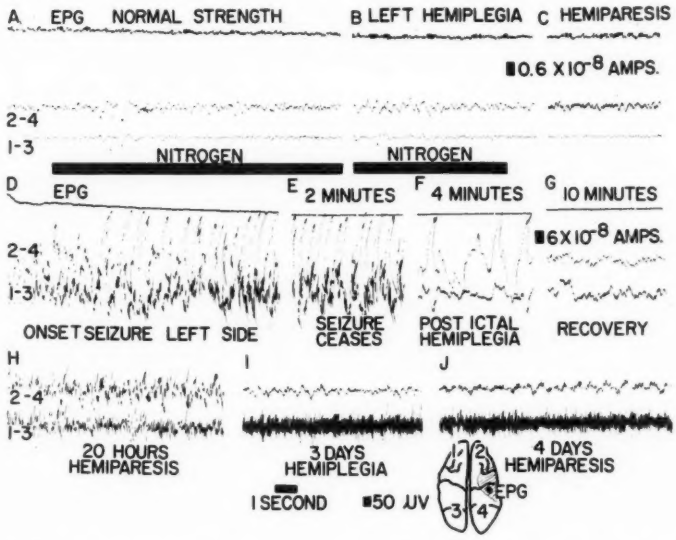

FIG. 6. A, B and C are 3 records from same animal made 7 wks. after occlusion of right middle cerebral artery to show production of transient hemiplegia by breathing 100 per cent nitrogen. (A) shows slow gradient of fall in oxygen tension (EPG) recorded from zone of infarction (see diagram of brain at bottom of figure). EEG from ischemic hemisphere (2–4) shows progressive slowing while EEG from left hemisphere (1–3) shows little change. (B) shows EEG and EPG after breathing nitrogen for 1 min. Left hemiplegia was now present. EPG was reduced from initial readings of 6 to 5.4×10–8 Amp. (C) 3 min. later EPG shows a rise with improvement in both EEG and degree of paralysis.

D, E, F and G are 4 samples of a continuous record from a monkey treated with heparin and Dicumarol whose right middle cerebral artery was occluded 2 days before, to show production of transient hemiplegia following a spontaneous localized left-sided seizure. Initial EPG reading was 6×10–8 Amp. but rapidly fell to 2×10–8 Amp. in record E at which time the seizure ceased. Oxygen tension remained low and posticta hemiplegia persisted when record F was made 4 min. later. Record G was made 10 min. later as animal recovered from hemiplegia.

H, I and J are 3 EEG records from another animal to show spontaneous fluctuation in functional defect caused by changes in collateral circulation. (H) 20 hrs. after occlusion; mild left hemiparesis was present. (I) 3 days after occlusion severe hemiplegia was present. (J) On the 4th day recovery was beginning.

brain swelling was so severe that the cortex became flattened against the window and cut off its own blood supply by compression of the pial vessels (Fig. 9B). If death resulted from brain swelling it occurred within the first 4 days when increased vascular permeability was at a maximum. The substance of the brain was seen to increase in volume beneath the window, resulting in flattening of the gyri and obliteration of the sulci. Diapedesis of red cells through the walls of vessels was pronounced and if trypan blue was injected intravenously 24–28 hours after the production of infarction the staining of the ischemic zone was greater than reported within the first 8 hours.[6]

Severe anemic infarction after occlusion of the middle cerebral artery

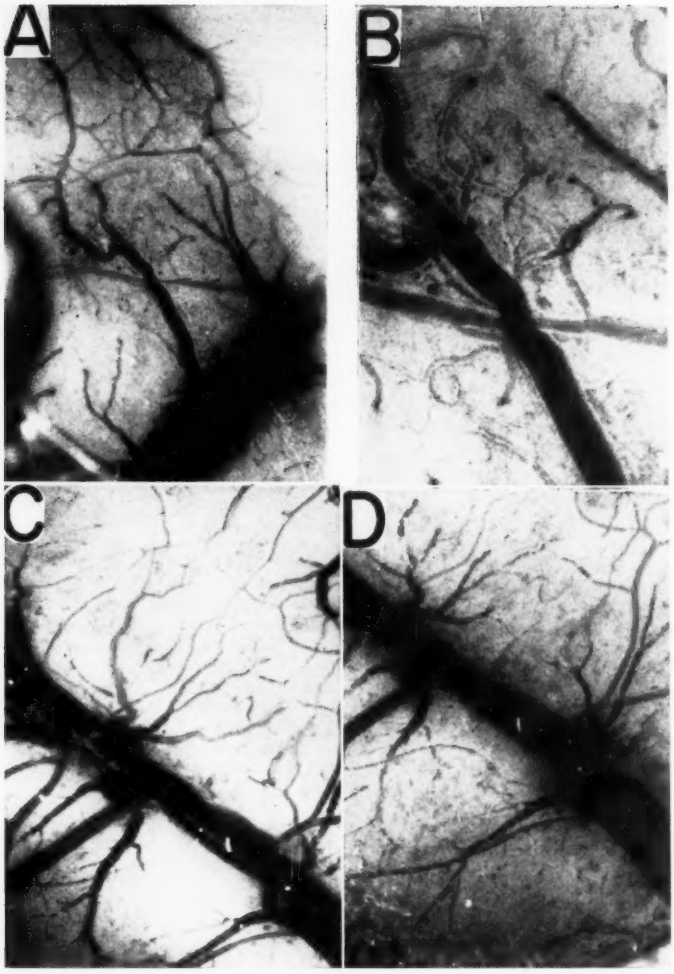

FIG. 7. Series of macrophotographs from the same animal.

(A) Macrophotograph taken at magnification of 18× to show collapse of pial vessels with segmentation, stasis and diapedesis in small venules 5 hrs. after occlusion of middle cerebral artery. Animal was stuporous at this time.

(B) High-power macrophotograph (54×) to show detail of collapsed pial artery. There is stasis in terminal arteriole, capillary and venule. The artery nicks the vein at point of crossing because of decreased intraluminal pressure. Capillary and venular networks are outlined by perivascular hemorrhages. Granular appearance of terminal venules is caused by clumping of red cells.

(C) Appearance of pial vessels 20 hrs. after occlusion to show stasis in terminal venules (×18). The animal showed severe flaccid hemiplegia.

(D) Appearance of pial vessels 24 hrs. after occlusion to show progressive stasis in some vessels and return of flow in others and an increase in perivascular hemorrhages. There are white thrombi in two of the veins (×18). The animal was drowsy with complete hemiplegia and hemisensory defect.

was produced regularly in the normotensive animal by placing clips on major anastomotic cortical vessels or by occluding the anterior cerebral or opposite middle cerebral artery (Fig. 8).

Delayed Resolution of Stasis. Stasis present in small vessels for periods up to 4 days regressed if the collateral blood flow improved after delay (Figs. 7 and 8). Such delayed increases in the collateral flow resulted in

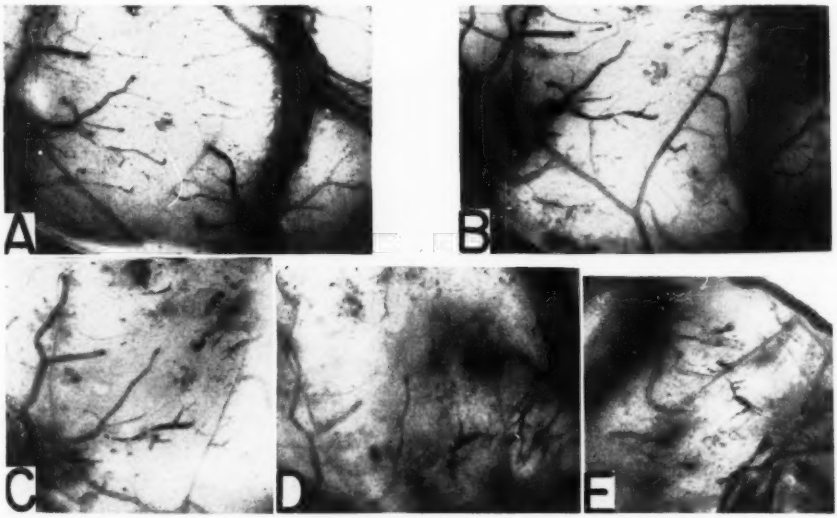

FIG. 8. Series of 5 macrophotographs (15×) of same area of cortex in a normotensive animal taken at different times following occlusion of right middle cerebral artery, the opposite middle cerebral artery having been occluded 7 days before.

(A) 2 hrs. after occlusion there is widespread segmentation and stasis in veins and venules. Animal was stuporous at this time.

(B) 1 day later. There is return of flow to many vessels with perivascular hemorrhages. Bilateral hemiparesis and left homonymous hemianopia were present and animal was drowsy.

(C) 2 days later. Flow continues to return to zones of stasis with further hemorrhage. Animal showed partial recovery from bilateral hemiplegia.

(D) 4 days later. Further return of circulation with more extensive perivascular hemorrhages. Bilateral hemiparesis continued to improve.

(E) 5 days later. Little stasis remains. Some of the old perivascular hemorrhages have been removed but fresh perivascular hemorrhages have also occurred. Vision had returned to left visual field and moderate spastic left hemiparesis persisted.

movement of the static clumps of blood into bordering zones of normal circulation where the clumps disintegrated and normal flow was resumed in the damaged vessel. The collateral flow may vary from day to day with intermittent stasis and resumption of flow in small vessels. Intermittent restoration of flow in static vessels resulted in progressive diapedesis and hemorrhage which produced extensive hemorrhagic infarction consequent upon anoxic damage to the walls of the vessels (Figs. 9 and 10B). Varying degrees of hemorrhagic infarction reflect the pattern of development of the collateral

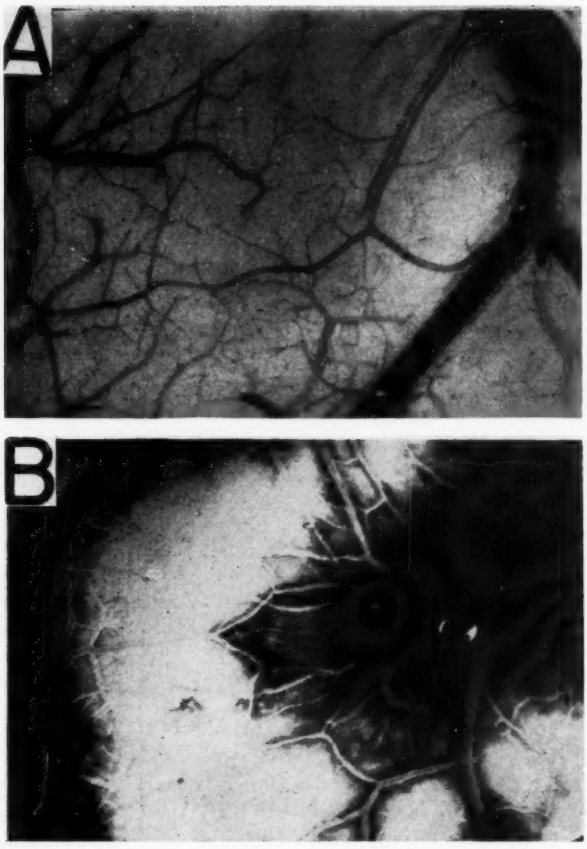

FIG. 9. Macrophotographs taken at magnification of 13×. (A) Massive ischemic infarct 45 min. after occlusion of middle cerebral artery, showing brain swelling and collapse of capillary network. The brain is pale and cyanotic and stasis is beginning in lower portion of field. (B) Massive hemorrhagic infarct 4 hrs. after rupture and occlusion of middle cerebral artery. Brain swelling is so severe that the pial vessels became collapsed and empty because of pressure against glass window. Animal remained in stupor, with dilated pupils and periodic respiration and died 18 hrs. later.

flow. Restoration of flow commonly resulted in recovery from the functional defect in spite of perivascular hemorrhage. Thus, commonly the hemorrhagic component of infarction progressed while function was improving. If the hemorrhagic infarction was severe enough, however, secondary worsening of the hemiplegia resulted because of brain hemorrhage and edema.[10,12] Spontaneous fluctuations in the collateral blood flow were common within the first 5 days and were closely correlated with intermittent changes in the hemiplegia (Figs. 6H, I, J, 7 and 8). After 14 days the collateral circulation became more stable, functional improvement was steady

and hemorrhages were gradually absorbed. Areas of necrosis were phagocytosed by white cells and the collateral circulation was supplemented by the regeneration of new vessels from the edge of the infarcted zone.

Provocation of Transient Ischemic Attacks. During the stage of recovery from the hemiparesis and for the longest period of observation in these experiments (9 weeks) the collateral circulation was barely able to supply the metabolic requirements of the ischemic zone in spite of any reduced metabolism resulting from ischemic damage. Factors that temporarily de-

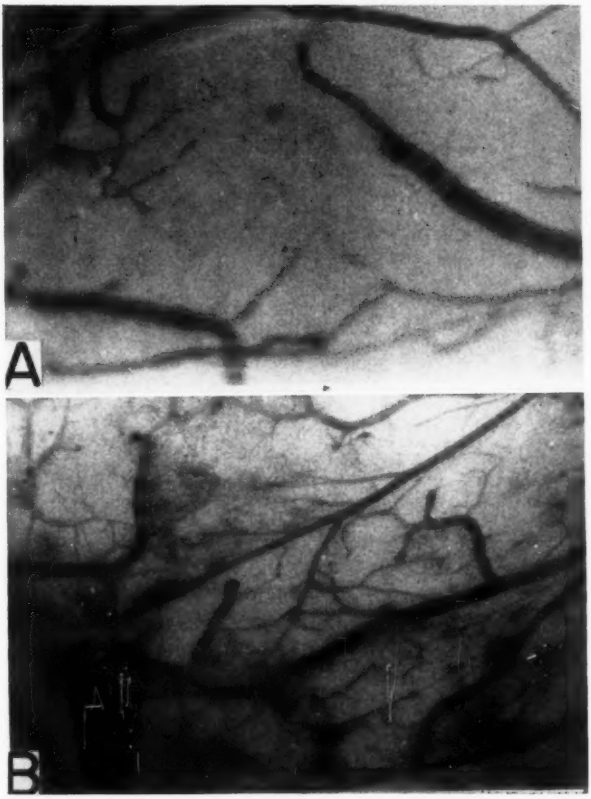

FIG. 10. To show absence of stasis and segmentation following occlusion of middle cerebral artery in 2 heparinized monkeys.

(A) Macrophotograph (135×) made 4 hrs. after occlusion of middle cerebral artery with blood pressure of 70 mm. Hg. There is cyanosis of terminal venule and capillary network is collapsed but there is not clumping or stasis of red blood cells. Severe hemiplegia was present. Clotting time 40 sec.

(B) Macrophotograph (56×) made 24 hrs. after occlusion of middle cerebral artery. Clotting time 45 sec. At time of occlusion systolic blood pressure fell to 65 mm. Hg, but when this photograph was made blood pressure had risen to 100 mm. Hg. In spite of marked perivenous hemorrhages no stasis occurred.

creased the collateral circulation or increased the local metabolic demand resulted in worsening of the functional defect. For example, repeated episodes of hemiplegia of 1–3 days' duration were induced by reduction of the blood pressure to 50–70 mm. of mercury for 10–20 minutes in animals that had recovered from occlusion of the middle cerebral artery. During these attacks, the pial circulation slowed with cyanosis of the veins and collapse of the vascular network, with prompt return of collateral flow as the blood pressure increased. The longer the hypotension was maintained (20 to 80 minutes) the more severe were the resultant functional defect and stasis of the pial vessels. For example, prolonged reduction in blood pressure below 65 mm. of mercury by the injection of 10 mg./kg. of hexamethonium or reduction of circulating blood volume produced regularly a severe hemiplegia associated with visible infarction of the cortex in the territory supplied by the occluded vessel (Figs. 3 and 4).

Anoxic anoxia produced by brief breathing of nitrogen or carbon monoxide also provoked a transient hemiplegia of 12–72 hours' duration in animals recovered from occlusion of the middle cerebral artery (Fig. 6A, B, C). Recording of oxygen tension from the infarcted zone during breathing of nitrogen showed a slower gradient of fall than is seen in normal cortex when 100 per cent nitrogen is breathed. In some instances reinfarction was produced by the anoxia. This was particularly true of healed infarcts of 2 to 5 weeks' duration when newly regenerated vessels are particularly prone to anoxic damage.

Postictal Paralysis. Four of the animals in this series of experiments were observed to have repeated localized seizures limited to the paretic side 2–21 days after occlusion of the middle cerebral artery. After the seizures subsided the degree of paralysis was worsened temporarily. In all instances there was some degree of hemorrhagic infarction visible beneath the window. Harvey and Rasmussen[13,14] reported similar spontaneous seizures in several of their animals recovering from occlusion of the middle cerebral artery. In 2 animals with a mild hemiparesis caused by occlusion of the middle cerebral artery spontaneous localized seizures occurred while recording the electroencephalogram and cortical oxygen tension from the ischemic zone. Fig. 6D is a record from one of these animals showing the onset of the seizure with the appearance of high voltage spikes in the electroencephalogram and clonic twitching of the left face and limbs. There was a rapid fall of cortical oxygen tension to near zero levels ($2\times10-8$ Amp.).[2] When the seizure subsided there developed complete flaccid hemiplegia of the left side and the electroencephalogram showed high voltage slowing. The cortical oxygen tension gradually increased over the ensuing 10 minutes, the electroencephalogram became more nearly normal, and strength of the left side was equal to that present prior to the seizure. In an additional animal focal seizures with consequent worsening of the hemiparesis were provoked by the administration of Metrazol by injection.

Effect of Administration of Anticoagulant Drugs. The administration of

heparin or Dicumarol prior to occlusion of the middle cerebral artery prevented the development of stasis or clumping of red cells within the ischemic zone in every instance (Fig. 10A, B). In one instance the systolic blood pressure was lowered to 20 mm. Hg without producing stasis. There was not any significant difference in the size of the infarcts in either the anticoagulated or control group but the survival rate was greater in the anticoagulated group and if recovery occurred it was frequently more rapid than in the control group. The infarcts in the anticoagulated group were more hemorrhagic than in the control group, similar to the observations reported by Whysnant[31] in 1957.

The production of transient ischemic attacks after recovery from the hemiplegia by anemically and chemically induced hypotension or by breathing of nitrogen was rendered more difficult in the anticoagulated group and the duration of the functional defect, if it could be provoked, was significantly shorter ($\frac{1}{2}$–3hours) than in the control group (1–3 days). Furthermore, in none of the anticoagulated group was stasis or infarction produced by hypotension or anoxia.

DISCUSSION

Occlusion of the main trunk of the middle cerebral artery in the monkey produces regularly a contralateral motor and sensory defect because of ischemic anoxia, the severity of which depends upon the functional adequacy of the collateral circulation derived principally from arterio-arterial anastomoses in the cortex. As observed by others[27] the severity of the hemiplegia bears a close relationship to the level of the systolic blood pressure at the time of occlusion; if it falls below 70 mm. Hg a severe hemiplegia with a persistent neurological defect follows regularly. Daily observations of the cortical vessels reveal that the collateral circulation fails if the blood pressure is not maintained, with resulting infarction of the cortex visible in the living animal.

If the blood pressure is well maintained the collateral circulation is barely sufficient to meet the metabolic demands necessary for survival of tissue within the first 8 hours and is not sufficient to maintain normal neuronal function, resulting in contralateral motor and sensory paralysis of variable degree. During this phase there is inadequate supply of oxygen as shown by the blue-black appearance of the veins and the low cortical oxygen tension, but the ischemic anoxia usually is not sufficient in degree to produce damage to the vascular endothelium with consequent intravascular clumping of red blood cells. This state of impaired circulation persists for 4 to 8 hours; thereafter, during the ensuing 24–48 hours there is a striking hyperemia of the previously ischemic zone caused by increased rate of flow in the cortical capillaries, many of which become visible for the first time. The entire vascular network is congested and the arterio-arterial anastomoses are widely dilated. At this stage, the dilated vessels can be caused to narrow in response to increase of blood pressure, for example by the intra-

venous injection of adrenalin (Fig. 5). This confirms the important role played by reduced intraluminal pressure in the dilatation of these arterial anastomoses.[20] The consequent rise in blood pressure results in rapid localized constriction of the dilated arterio-arterial communications, later followed by dilatation again as the blood pressure returns to normal levels.

If the collateral circulation fails because of a fall in blood pressure or the application of clips to important collateral vessels, the ischemic anoxia is of sufficient degree to damage the endothelium of vessels, resulting in intravascular stasis beginning at small points in the capillary network with consequent increase in local cerebrovascular resistance.[6] At this time the cortical oxygen is near zero levels. The endothelial damage results in the extravasation of the fluid constituents of the blood resulting in edema of the brain visible through the window. The increased vascular permeability reaches a maximum 48–72 hours after occlusion and the intravenous injection of trypan blue at this time results in a localized, intense blue staining of the brain substance with the dye. The increased vascular permeability also results in diapedesis of red cells and if the vascular damage is severe enough rupture occurs with the development of perivascular ball-hemorrhages (Fig. 3C). Stasis frequently continues to progress until the fourth day, although the process may be intermittent because of fluctuations in an unstable collateral circulation. Improved collateral blood flow results in slow movement of previously static clumps of red cells to zones of normal flow where the clumps disintegrate and normal flow is restored, frequently resulting in vascular rupture and hemorrhage. If stasis persists beyond the fourth day the vessel and its static content of red cells are phagocytosed by white cells.

After the second day patchy infarction of the cortex with softening becomes recognizable in the living animal. Microscopic examination of cortex shows that clouds of white cells derived from remaining intact vessels invade the area of necrosis (Fig. 3D). The clouds of white cells rapidly ingest necrotic brain and extravasated red cells and assume a granular yellow or golden-brown appearance. The stuffed histiocytes then migrate back to vessels and re-enter the blood stream. Detailed descriptions of changes in the properties of circulating elements in the blood occurring in the ischemic zone have been published recently[17] and neuropathological studies will be reported subsequently.[18] Commonly fluctuations in the severity of the hemiparesis and the degree of slow activity present in the electroencephalogram correspond to variations in the collateral circulation.

Proliferation of capillaries at the edge of the infarct progresses rapidly and their differentiation into arterioles and venules provide additional collateral circulation. For the first week after their appearance these new vessels are friable and rupture easily with consequent hemorrhage. The revascularized edge of the infarct has a high oxygen tension whereas the necrotic center usually has a low oxygen tension.

After the second week the collateral circulation becomes more stable. Up to this point it is clear from the appearance of the brain, the measure-

ments of oxygen tension and the recovery of the electroencephalogram that much of the functional improvement is the result of restoration of neuronal function temporarily impaired but not destroyed by ischemia. Thereafter, any further slow improvement is presumably ascribable to reorganization of function within a permanently damaged nervous system.

The transient ischemic attacks provoked in the recovered animal by anoxic anoxia, by reduction of blood pressure and by epileptic attacks[9] appear to have a common mechanism in that they all tend to decompensate a tenuous balance between the supply of oxygen derived from the collateral circulation and the metabolic requirement of the ischemic area. Any measure that temporarily decreases the collateral blood flow or increases the local metabolic requirement results in failure of the local supply to meet the demand of the tissue, with resulting temporary functional impairment. These data amplify and confirm the experiments of Corday *et al.*[1] concerning the role of hypotension in the production of transient ischemic attacks.

These authors produced electroencephalographic changes in the distribution of an occluded internal carotid artery in the monkey by lowering the blood pressure.

The Nature and Significance of Hemorrhagic Infarction. Direct observations reported here show that hemorrhagic infarction is the result of re-establishment of blood flow in vessels damaged by ischemic anoxia and that this process of hemorrhagic infarction may progress while function improves. Thus, hemorrhagic infarction indicates that at some time there has been improvement in the collateral circulation whereas a "pale" infarct represents failure of the collateral circulation. Microscopic observation in the living brain emphasizes that the division between "pale" and hemorrhagic infarcts is an arbitrary one since microscopic hemorrhages are common in "pale" infarcts.

Thus, anticoagulant drugs that prevent stasis increase the return of blood to ischemic zones via the collateral circulation. If the zone is already infarcted there results a greater degree of hemorrhagic infarction without functional improvement. If the ischemia is less severe, for example, in the experimental production of transient ischemic attacks by hypotension or anoxia, the prevention of stasis by anticoagulant drugs improves collateral circulation. As a result, transient failure of the collateral circulation is harder to provoke in the anticoagulated preparation but, once provoked, recovery is more rapid than in similar preparations not treated with anticoagulant drugs.

SUMMARY

Occlusion of the middle cerebral artery in the monkey results in a functional deficit ranging from transient hemiparesis with recovery to severe and persistent hemiplegia. Lowering the systolic blood pressure below 70 mm. Hg or occlusion of principal collateral vessels in addition to occluding the middle cerebral artery consistently produces severe hemiplegia. Ac-

companying vascular changes are visible through a skull window placed in the distribution of the middle cerebral artery. Transient hemiplegia is accompanied by cyanosis of the pial vessels with slowing of flow but rapid restoration of pial circulation by collateral vessels. Severe hemiplegia results when the collateral circulation fails and there develop clumping, segmentation and stasis of red cells within vessels. Ischemic anoxia produces damage to the endothelium of the vessels and edema. Restoration of blood flow to damaged vessels results in perivascular hemorrhages. Heparin and Dicumarol prevent stasis in the collateral vessels.

In the animal recovered from occlusion of the middle cerebral artery transient hemiplegia may be provoked either by reduction of the systolic blood pressure, resulting in temporary collapse of the collateral circulation, or by anoxic anoxia or by increased neuronal consumption of oxygen consequent upon convulsive activity originating in the ischemic zone. Heparin and Dicumarol render production of transient hemiplegia by these methods more difficult.

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RECENT TRENDS IN NEUROSURGERY*

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A review of current neurosurgical literature from all over the world emphasizes the fact that neurosurgery as a specialty is no longer limited to the universities or large metropolitan areas. Articles on the subject appear from almost every country on the globe, and many of these countries, however small, have one or more journals devoted, if not to neurosurgery alone, then at least to the more general field of neurology. Whereas by far the major portion of neurosurgical practice in the United States is in the hands of trained neurosurgeons, this still is not the condition found in many other countries, though it is obvious that even in those places there is a steady and fast-growing tendency to the development of neurosurgery as a special surgical art. This review, covering the published material over the last several months (and not particularly concerned with case reports), attempts to indicate the general trends in neurosurgery especially outside the United States, but including certain pertinent material from this country.

HEAD

The preoperative localization of tumors by mechanical means has been of general interest for several years, and recently Soille,[74] in a study of 43 patients with verified intracranial tumors located behind the postcentral gyrus, noted that arteriograms showed on side view a spreading of the terminal branches of the sylvian artery, increased tortuosity, and an elevation of its initial part. The anterior curl of the sylvian artery was pushed downward, but the syphon was never stretched, and usually was flattened. On anteroposterior view the most striking sign was a lowering of the first part of the artery, while the middle portion of the anterior cerebral artery was pushed toward the unaffected hemisphere. The exact type of tumor and its particular location in the posterior half of the hemisphere produced varying percentages of these changes. Another report[72] is an analysis of the electroencephalograms of 100 patients with unilateral supratentorial tumors, studied from waking to sleep states, compared to the operative findings. Tumors causing a persistence of the slow-wave focus into sleep with suppression of sleep potentials were the most superficial with respect to convexity of the brain. Tumors causing only a persistence of the slow-wave focus were likewise superficial, but deeper than the former. Tumors causing a disappearance of the delta focus and a relative lack of distortion of sleep patterns were the deepest and were more than 3 cm. from the convexity of the brain.

After a study of 250 histologically verified brain tumors, Götze and Kubicki[30] felt that the degree of malignancy could be prognosticated by a comparison of angiograms, pneumoencephalograms, and electroencephalographic records. *Gross* dislocations seen in angiograms and pneumoencephalograms with *minor* electroencephalographic changes indicated a benign tumor in 93 per cent of the patients. *Minor* dislocations in the radiographs with *minor* electroencephalographic changes indicated a benign lesion in 89 per cent of the patients. *Minor* dislocations in the

* Prepared at the request of the Editorial Board of the *Journal of Neurosurgery.*

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radiographs with *major* electroencephalographic changes were found to indicate malignancy in 87 per cent of the cases. *Gross* dislocations with *major* electroencephalographic changes showed a malignancy rate of 72 per cent.

There is much current interest in European literature, particularly the French,[3] having to do with the diagnostic difficulties of posterior fossa, particularly angle, tumors, with much emphasis on the reliability of the mechanical methods of diagnosis (especially air studies). Spillane *et al.*[75] have stressed the variety of actual anatomical changes to be found in the region of the foramen magnum, as revealed by surgical exposure, in contrast to what may have been revealed by preoperative radiographic studies. This paper, particularly well illustrated and with an excellent bibliography, is a good review of the clinical, radiologic, and operative characteristics of a region known for trickiness. Nachtwey[58] insists that if there is any reason to believe that suboccipital or upper cervical pathology of any sort exists, clinically or radiologically, then suboccipital puncture or needling of any sort must never be done. Abnormal vascularity and displacement of vessels, the brain stem, and upper cord are possibilities that make such puncture dangerous.

Interest continues in certain tumor groups, notably the benign ones, and particularly the meningiomas,[18,34,60,88] epidermoids,[49] chordomas,[50] and certain tumors of childhood,[11] and in those conditions having to do with optic atrophy *per se* and with unilateral exophthalmos.[13,53,64,82] Tumors of the hypophysis are discussed widely, ranging from methods of technique for removal, such as the subfrontal, extradural, extracapsular removal advocated by Freshwater *et al.*,[25] to the transphenoidal operation so persistently, and be it admitted impressively, urged by Hirsch[39] in his review of 413 patients so operated upon, in conjunction with the use of radium implants. Horrax[41] was a strong advocate of the method of treating such tumors by deep radiation, and he believed that with modern improved apparatus and technique operations upon the hypophysis, with its attendant dangers, can often be avoided and the over-all results of treatment improved. Londoño[54] reported some very interesting results of his electroencephalographic studies of 22 patients with pituitary adenoma and 5 with craniopharyngioma. He showed the changes in electrocortical rhythms to be of three types: generalized disorder with but little modification of frequency; the appearance of theta or delta rhythms; and the appearance of fast rhythms. He believed that these changes are caused by the effect of the tumor on the hypothalamus, and that this involvement activates the cerebral cortex through the anterior and dorsomedial thalamic nuclei and the reticular substance of the brain stem.

Wertheimer and Corradi[84] reported a group of 18 patients with craniopharyngioma (12 men, 6 women) averaging 49 years of age, but ranging from 40 to over 70 years of age. They pointed out that whereas the radiographic and histologic appearances of the tumor present nothing unusual, the clinical signs are different in the older age group from those in children. In the younger patient the main clinical sign may be greatly increased intracranial pressure; in the older patient this is less often true, but instead one finds signs of meningeal irritation, decreasing vision, somnolence, weakness, loss of libido, and amenorrhea. They feel that complete removal of the tumor is desirable but rarely possible, and that in the older patient radical surgery so often leads to death that the surgeon may have to content himself with only drainage of the cyst. Two of their patients were found to have craniopharyngiomas that had undergone transformation into an epithelial type of meningioma, and they believe that pre-operative differential diagnosis must consider

meningioma, aneurysm of the syphon, chordoma, teratoma, and epidermoid tumor. Tiberin *et al.*[78] also feel that the clinical symptoms of craniopharyngiomas are less "typical" in adults than in children. Their adult patients showed striking mental changes, and it is pointed out that in the older patient radiographic and visual changes may be so slight, and mental changes so marked, that a mistaken diagnosis of cerebral arteriosclerosis may be made. They emphasized the point that such patients are incapable of meeting the stress of intracranial surgery. Cortisone in one form or another is used widely in an attempt to meet such operative stress, in patients of all ages and for surgery both in the hypothalamic region and elsewhere intracranially. Jenkner[46] has reported upon the use of a new drug, an anticholinergic, —10-(2-diethylaminopropyl) phenothiazine methochloride (furnished by G. D. Searle & Co.), as a treatment for postoperative and post-traumatic cerebral edema that leads to coma, hyperthermia, and other such signs. He has used it intravenously, intrathecally, intraventricularly, and topically at the site of operation, and it was particularly effective in the coma of craniocerebral trauma. His studies did not reveal whether the high content of acetylcholine in the cerebrospinal fluid was caused by a primary disturbance of cell function, or by a change in cell permeability, or still other factors that lead to the appearance of the substance. He advocates the use of this drug as a *preventive* measure, and as better than fighting the situation, once developed, by such means as hibernation, ganglionic blocking and ACTH. He has found this new drug more effective in cerebral than in posterior fossa lesions.

One sometimes finds the opinion expressed by neuropathologists that neurosurgeons would do well to leave the subject of pathology to pathologists, and content themselves with the practice of neurosurgery. There is something to be said for this attitude, particularly when reports, such as that of Arseni,[6] indicate the need of a common language in classification of tumors, a subject which admittedly has been muddled by some neurosurgeons. Nothing current has been found to improve upon the old standby of Bailey and Cushing, but it is true that the recent work of Zülch[90] does shed new light without upsetting time-tested classifications.

Craniocerebral injury continues to hold its interest to essayists,[16,17,32,81,85] and probably will continue to do so in this increasingly mechanical world. Most of the reports simply reiterate the well-tested neurosurgical principles to be used, and contain little that is actually original. The conflict in Algeria has led French neurosurgeons[21] there to re-emphasize the importance of early evacuation of the head-wounded patient to the rear where *one* definitive operation can be done with surgical completeness. Again, as was learned in World War II, they have noted the importance of removing large metallic fragments and *all* bone fragments, of leaving smaller metallic fragments alone, and of using antibiotics freely.

There are three recent reports of interest having to do with the treatment of hydrocephalus. Lazorthes *et al.*[52] have described a method of transcallosal ventriculocisternostomy which sounds impressive. It is useful in adults as well as children, and has the great advantage that the patient's growth or movements of the head do not affect its function. A small plastic tube is placed downward through the corpus callosum into the ventricle, resting near the interventricular septum, and draining superiorly into the pericallosal cistern. The article describes the apparatus and surgical technique in detail. Sikkens[71] has improvised a tube with ball valves to prevent the reflux of blood in his ventriculojugular shunt (an old method, actually, and first used, without success, by Gärtner in 1895). If it is as successful as he claims it to be in the treatment of any type of hydrocephalus (and it would seem that his

enthusiasm might require a bit of tempering), then it is a great improvement over such methods as the Torkildsen procedure or drainage into a ureter, the pleural or peritoneal spaces, or into other areas where the cerebrospinal fluid contents are lost to the body. Feld,[23] on the other hand, feels that coagulation of the chorioid plexuses, usually only on one side, by direct ventriculoscopy in nonobstructive hydrocephalus is to be preferred to any method of mechanical drainage of the fluid. He gets his indication for or against operation by first determining by pneumoencephalography the "corticoventricular coefficient," that is, whether or not there is enough cortical substance in existence to warrant any attempt to save the patient. In short, there seems to be little new in the realm of treatment of hydrocephalus.

The major source of reports having to do with destructive lesions of the basal ganglia in the treatment of Parkinsonism-like syndromes still continues to be the neurosurgeons of the United States, the focal point seemingly the symposia of the St. Barnabas Hospital, New York. Ultrasonic irradiation of the ansa lenticularis,[61] stereotactic technique employing either chemical injection or electrocoagulation, and direct surgical attack via the blood supply are some of the various methods in use of this rapidly expanding new field. Gildenberg,[29] using cats in a study of the variability of subcortical lesions produced by several procedures, found that direct current applied by means of the monopolar electrode offers less variability than any other method. Radio-frequency coagulation produces a lesion along the canal of puncture, and alcohol produces erratic, unpredictable lesions. His excellent article stresses the importance of several basic factors in the apparatus to be used. Housepian and Guzman-Lopez[42] have shown that fluids injected, however slowly, into the depths of the brain produce necrotizing lesions of uncertain size and shape, and that the fluids leak out along the needle during injection and along the path of puncture after withdrawal of the needle. Their warning is strong against the use of potentially toxic materials for such injections. Hromada[44] has studied in detail 52 human brains for the origin, course, branching characteristics, area of supply, and collaterals of the anterior chorioidal artery, and he has found its anatomy to be of such a constant and predictable nature that surgical identification is almost always assured for accurate surgical occlusion of the artery. In no case did he find the artery lacking. It was in duplicate in 1.9 per cent, and in 0.9 per cent of the cases it arose from the posterior communicating artery instead of from its usual origin on the internal carotid or the first part of the middle cerebral artery. As if to deflate such surgical enthusiasm, Italian workers[28] have recently shown what can be done medically with safe and lasting effect in the treatment of Parkinsonism. In a group of 64 patients they used a new compound, "10870" (3-phenyl-3 (beta-diethylaminoethyl)-2-6-dioxypeperidino-hydrochloride), and after 60 days of continuous treatment found that the drug was well tolerated, produced few ill side effects, and gave clinically gratifying results, particularly in relieving hypertonia, tremor, impairment of speech, vegetative changes such as drooling and sweating, and psychic disturbances. The drug worked less well for the postencephalitic type of patient, and combined with Ritalin or Reserpine it was less effective than when used alone.

Interest in the problems of vascular lesions within the brain seems to be increasing rapidly, especially in Europe.[4,36,67,83] Arnulf's[4] recent publication is particularly informative of the anatomy, pathology, and clinical considerations having to do with the carotid arteries. Sastrasin's[69] article is a good résumé of current thinking on the subject of carotid thrombosis, containing a clear discussion of the clinical syndrome and an excellent bibliography. He feels that the lesion can be diagnosed

with certainty only by arteriography, though admittedly it is an especially dangerous procedure in this lesion. He finds that most carotid thromboses occur on the left side, and usually at the carotid bifurcation in the neck or at the syphon, because of the predilection for atheromatous plaques to form at these sites. His outlook on the results of either medical or surgical treatment is definitely more pessimistic than that of Lyons and Galbraith.[55] Törmä and Troupp[79] found 31 instances of carotid thrombosis in approximately 3000 angiograms, or about 1 per cent. The lesion was more common in males than females, and more often on the left side. They feel that the single most important factor in the genesis of the thrombus is arteriosclerosis, which they found in the carotid arteries of male subjects as young as 20 years of age. The thrombosis may also be the result of local trauma or a lesion such as a tumor. They find little hope in attempts at thrombectomy, that most other attempts at surgical correction are disappointing, and that if the lesion is not immediately fatal it will almost certainly leave the patient with a crippling cerebral vascular insufficiency.

In cases of spontaneous intracerebral hemorrhage Gomensoro and Arana Iñiguez[31] depend greatly upon arteriographic findings to determine whether or not the lesion can be treated surgically. In the acute state, requiring immediate relief, open operation (osteoplastic craniotomy) should be done with the evacuation of the clot under direct vision, though the mortality in such patients is bound to be much higher than in those whose condition permits a wait of at least 7 days. The ideal time for surgery seems to be approximately 15 days after onset of symptoms, when the patient is in a more or less stable condition. The fact that the patient is often arteriosclerotic adds to the unpredictability of the outcome. Howell's[43] series of 33 patients with massive spontaneous intracerebral hemorrhage showed an operative mortality of 21 per cent: 5 per cent for a nonhypertensive group of 19 patients and 43 per cent in the hypertensive group of 14 patients. Of the survivors, epilepsy developed in 35 per cent. He advises early operation (craniotomy) and stresses the dangers and inadequacies of trying to aspirate the clot through a burr hole.

The mass of reports having to do with pathology, operative methods, instruments and drugs, and survival and mortality statistics of intracranial aneurysms continues to flood the literature.[8,22,33,36,67,68,73] Much of it comes from Europe. Huhn[45] has shown the value of arteriography not only in the diagnosis of aneurysms, but also in locating thromboses of venous structures intracranially. He points out the importance of such venous lesions arising as the result of delivery, trauma, operation, degenerative processes, general circulatory failure, thrombophlebitis elsewhere in the body, and acute or chronic infection in the upper respiratory tree. His article reflects the increasing European interest in the technique and interpretation of angiograms. As an example of the enthusiasm shown in some quarters for panarteriography, Zaclis and de Almeida[89] have reported what must certainly be a dangerous as well as intriguing procedure! They noted that the vertebrobasilar system would sometimes fill during carotid arteriography, and they thought it could be explained by a sudden marked drop in blood pressure and blood flow caused by a reflex elicited in a hypersensitive carotid sinus. Feeling that the use of hypotensive drugs for arteriography is unsafe, they have used the method of Boerma and Blickman (1955) for the reduction of intrathoracic pressure to 60 cm. of water. The blood pressure also is then quite low, and at that point arterial pulsation ceases. Fifteen cc. of 35 per cent Nosylan are injected into one carotid artery at this time. In each of their 20 patients all the intracranial branches of the ispilateral carotid and vertebro-

basilar systems have been visualized. In most patients even these arteries on the *opposite* side were visualized. On the restoration of normal intrathoracic pressure, the blood pressure and flow are restored and the contrast medium is washed from the brain. The procedure seems a bit heroic in the light of the fact that apparently safer, if more cumbersome, methods for demonstrating the main parts of the cerebral vascular tree can be performed, but they report no accidents. They are looking forward to being able to report their results on the venous phases of panarteriography.

In the surgical attack upon an intracranial aneurysm, Gibbs[27] uses a special clamp for the temporary atraumatic proximal occlusion of a major cerebral vessel from which the lesion arises. With the clamp in place, the aneurysm may with certainty be ligated at its neck, opened, and its contents removed manually. By thus cleaning out the interior of the aneurysm it can be determined if all leakage has stopped, and furthermore there can be no chance of some of the clot passing on into the distal circulation, causing a postoperative hemiplegia which is often blamed on "postoperative spasm." Certain French surgeons[35] have been concerned with the treatment of those intracranial aneurysms that do not have a neck that can be clipped or ligated, or that are so located on a major vessel that they cannot be isolated by clipping that vessel. Into such an aneurysm they insert a fine bipolar electrode, coagulating it and its contents. When solidification and dessication have been obtained, the wire electrodes are clipped off short at the exterior of the aneurysm, and the wire tips are left undisturbed in the lesion. They report one such experience, with the patient symptom-free after 11 months, and with arteriographic proof of destruction of the aneurysm.

In 2786 consecutive autopsies, Chason and Hindman[15] found 137 instances (4.9 per cent) of berry aneurysm of the circle of Willis. Theirs was a most careful record, "doubtful" cases not having been included. The greatest incidence of the lesion was found in subjects aged 61–70 years. In these 137 brains, 196 aneurysms were found, 2 patients even having 5 such lesions. They point out that most reports indicate an incidence of from 0.5 per cent to 1.5 per cent berry aneurysms of the circle of Willis.

The surgical treatment of epilepsy is increasing in scope, be the disease the result of trauma, tumor, or the more evasive "temporal lobe focus."[38] Nittner[63] had his best results when the epileptogenic foci were in either the frontal or occipital lobes, and he always limited his surgery to the actual boundary of the visualized lesion. His most successful treatment was in cases of infantile lesions of the brain when he could remove a cyst or even perform hemispherectomy. Achslogh *et al.*[1] found that in a group of 62 patients with supratentorial angiomas, 36 had epilepsy and of these 23 showed focal fits. Ordinarily these focal fits located the angioma correctly, but in any event the electroencephalogram was very consistent in locating all the angiomas. Ligature of the carotid artery did not improve the epilepsy, and removal of the lesion improved the epilepsy in but one-half the patients so treated. Best results were obtained when the lesions were in the right frontal or temporal lobes, where wide resection could be carried out. Röttgen and Müller[66] feel that when as after any open head injury or any traumatic loss of continuity of brain substance, a connective-tissue scar appears, that scar should be resected as soon as possible regardless of any apparent effects of treatment with drugs. When a widespread glial proliferation occurs, as after closed head injury, surgery should be undertaken only when drugs have failed to control the seizures, when symptoms are very severe, and when the surgical resection can be guided by the electroencephalogram and electrocardiogram.

Maspes *et al.*[56] operated upon a group of 36 epileptic patients with nontumoral temporal lobe electroencephalographic foci. The principal etiologic factor in all 36 patients was closed brain injury, old foci of childhood meningo-encephalitis, and obstetrical trauma. They were unable to identify any special type of seizure for each anatomic type of lesion (cortical sclerosis, superficial meningocortical scars, deep temporal lobe sclerosis), but psychomotor phenomena and neurovegetative components of fits were noted in patients with all types of lesions. Thomalske and Woringer[77] have published a detailed discussion of focal epilepsy, including the anatomy of the temporal lobe and rhinencephalon, the normal physiology of these structures and their pathology in focal epilepsy, and the surgical treatment of such epilepsy, including hemispherectomy. They point out that surgical results, at first very encouraging, have a way of becoming lost, if the surgeon will but follow his patients and study them. These men are impressed by the value of hemispherectomy, the indications for it being infantile hemiplegia, medically uncontrolled focal epilepsy, or severe deterioration of character in the uncontrolled case. Among the many contraindications for such radical surgery they list: symptoms of pathologic change in the opposite hemisphere, idiocy, only a slight pre-existing hemiparesis, generally poor physical condition, or evidence of any atrophy of the structures of the posterior fossa. Umbach[80] is impressed by the usefulness and safety of stereotactic electrocoagulation of the basal ganglia in the treatment of certain types of epilepsy. Instead of temporal lobectomy, the fornix was interrupted in 4 patients, near the anterior commissure (2 unilaterally, 2 bilaterally). Unilateral coagulation of the fornix is recommended especially when the focus of epilepsy is in the dominant hemisphere. He urges that because the method is simple and accurate it should at least be tried before open operation with resection is employed. In a preliminary paper Wycis *et al.*[87] reported the effect of pallidotomy, or a combined lesion of pallidoamygdalotomy, for the relief of major and minor convulsions in patients in whom anticonvulsive medication has been ineffective and in whom seizure discharges can be demonstrated in the basal ganglia. Accurately placed lesions of desired size, produced by their stereoencephalatome, have shown lasting results. Their findings indicate an influence produced by the lesions not only upon the peripheral muscular manifestations of centrifugal impulses passing through the basal ganglia, but also upon subcortical epileptogenic foci affecting the functional state of the cortex.

SPINAL CORD

The spinal cord continues to arouse much less interest, both as to pathology and physiology, than do the intracranial contents, and much of the literature concerning the cord has to do with case histories. Brihaye and Lorthioir[12] reported a severe allergic reaction to 2.5 cm. of methylene blue injected intrathecally in a 53-year-old man who was being studied for a cerebrospinal fluid rhinorrhea. There was no difficulty in doing the lumbar puncture, but almost immediately upon injection of the dye there was motor, sensory, and sphincteric loss, persistent and practically complete. There were never any "meningeal signs." They urge the use of testing for tolerance whenever methylene blue is to be used in the cerebrospinal fluid system. Netsky[59] observed a patient suffering from multiple meningiomas of the spinal cord, and later from arachnoidal fibrosis and syringomyelia. At autopsy many foci of meningiomas were found epi- and subdurally. He believes that the tumors, together with the trauma of several laminectomies that had been done, caused a proliferation of connective tissue in the leptomeninges, leading to infarction of the cord and

subsequent formation of syrinx. Finneson *et al.*[24] reported the history of a 64-year-old woman, with known Paget's disease, who exhibited the syndrome of acute compression of the cauda equina. Laminectomy revealed an osteogenic sarcoma arising in the lumbar vertebrae involved in the osteitis deformans. Berthold[10] has pointed out that spinal epidural granulomas may cause a transverse lesion of the cord in much the same manner as does trauma—by compression which interrupts circulation of blood and cerebrospinal fluid. These lesions may be the result of tuberculosis, syphilis or so-called nonspecific infectious processes, and suppuration from them can extend into retroperitoneal and retropleural spaces. The term pachymeningitis in such patients should be dropped. Tarlov's[76] excellent monograph is by now familiar to all American neurosurgeons.

In a study of pain in postcordotomy patients, King[48] used stimulation of skin, direct stimulation of nerves, and nitrous-oxide inhalation of from 20 per cent to 60 per cent concentration. It is the author's opinion that late gliosis, or injured though not completely interrupted spinothalamic pathways, may be the cause of eventual return of some sensory power in the area supposedly desensitized, but he also postulates as probable the existence of a polysynaptic relay pathway for painful stimuli in man, aside from the spinothalamic system. More along this same general theme may be found in the paper by Pollock *et al.*[65] The monograph published under the direction of Alajouanine[2] is a most comprehensive treatise on the subject of pain, its origins, and its treatment.

There continues to be a trickle of reports concerning that old favorite, the herniated intervertebral disc. Favorite subtopics continue to be the value of myelography and to-fuse-or-not-to-fuse. Decker and Shapiro[19] found that the most reliable means of accurate diagnosis of the herniated disc was indeed myelography, it being much more dependable in their hands than a knowledge of the motor, sensory, and reflex changes (all of which they found, as many others like them have, to be confusingly inconsistent). Neither did they place any dependence on radiographic demonstration of narrowing of the interspaces. Spinal fusion was done in their 230 patients only when spondylolisthesis was present (3 per cent), and 92 per cent of their patients stated that they were benefited by surgery. Koskinen,[51] in a well documented series of 104 patients with herniated discs, has made a thorough report of the clinical histories with emphasis on the stage-by-stage development of the final clinical syndrome. His patients benefited by good follow-up study, and he found that 87 per cent of the patients were cured of their sciatica by surgical removal of the protruded disc, that 54 per cent were completely fit to return to work, and that 41 per cent could work with restrictions. It has of course long been apparent that a proper understanding of the problem of herniated discs can come only through carefully controlled selection of patients and common-sense surgical care, together with an impartial judgment of results in long follow-up studies in significantly large series of patients.

NERVES

Scanning the neurosurgical literature reveals a great paucity of interest in peripheral nerves, the latest publication of significance being the Veterans Administration Medical Monograph on peripheral nerve regeneration which appeared in June 1956.[86] Kettel[47] has described an ingenious method for repair of the facial nerve in traumatic facial palsy. In lesions of that nerve within the temporal bone, the facial nerve is first exposed intracranially, severed, and sutured to a graft (sural cutaneous)

at its proximal stump. The distal end of the graft is then marked with a silver clip and is brought out through the craniectomy and tunneled under the sternocleidomastoid and splenius capitis muscles, below the tip of the mastoid. After 90 days to allow for growth of nerve fibers into the graft, roentgen-ray localization of the clip is done and the distal end of the graft is brought forward. The facial nerve in the face is severed and the graft is sutured to the distal stump. In 4 patients there was 80 per cent recovery in 2 and 50 per cent recovery in the other 2. This operation is preferable to the intrapetrous graft technique of Ballance and Duell, especially if the lesion in the facial nerve is proximal to the geniculate ganglion or if the temporal bone is destroyed. Mendelsohn *et al.*[57] performed cervical myelography in 11 patients with stretch injury of the brachial plexus, and in 7 of them they demonstrated good evidence of avulsion of the root. They feel that such study is valuable in the avoidance of useless exploration of the brachial plexus following such injury, and that it leads to the early performance of reconstructive surgery.

AUTONOMIC NERVOUS SYSTEM

Now that improved means of medical management of hypertension have dulled neurosurgical interest (except in extreme cases) in that disease, there is little to be found in either American or foreign literature dealing with the neurosurgical aspects of the autonomic nervous system. However, Arnulf,[4] faithful protégé of René Leriche, has reviewed very well the possibilities of surgery of the sympathetic nervous pathways so far as the intracranial vascular tree is concerned.

INFECTION

American neurosurgeons are less concerned with the surgical aspects of infection of the nervous system than are those surgeons in other countries where especially tuberculosis and hydatid disease are more common. The Rumanian neurosurgeons, Arseni and Samitca,[7] have described the life cycle of *Taenia echinococcus* in an excellent publication which includes a lucid clinical discussion of the disease. Of 2,226 intracranial space-occupying lesions operated upon in a 20-year period, 36 (1.6 per cent) were cranial or intracranial hydatid lesions. Their patients usually had eosinophilia; the complement-fixation test was rarely positive; the electroencephalogram was not found reliable for localization; and no calcification of the cyst wall was found on roentgen-ray study of any patient. Twenty-eight per cent of their patients had mental disturbances. They found that ventriculography was the safest and most dependable method of localizing the lesion, and that surgical resection of the cyst through an osteoplastic craniotomy and cortical incision was a safe and effective method of treatment. Very often, after cortical incision over the cyst, the jugular vein can be compressed and the patient can be asked to "cough out" the cyst, intact. Approximately 90 per cent of the cysts are found to be sterile, but if one should rupture during removal no chemical of any kind should be used locally. The over-all results of surgery for this infection are very good. Arseni[5] also has furnished a good report of his experience with the treatment of tuberculoma of the central nervous system. In a series of 2,000 intracranial tumors, 180 instances of tuberculoma were found. Eight of these patients also had tuberculoma of the spinal cord. As treatment he recommends the surgical excision of the lesion, with care against contamination of the cerebrospinal fluid spaces. If all the lesion is not surgically resectable, the remaining portion should be thoroughly coagulated. The results of surgery are better in the cerebrum than in the cerebellum, and intramedullary lesions of the cord offer

a poor prognosis. Arseni feels that streptomycin should never be given intraventricularly, intrathecally or applied locally in the bed of the lesion. He gives it intramuscularly in daily doses of 1 gm. from the day of operation, for a total of 70 gm. to 90 gm. Isoniazid and PAS are given by mouth for 2 months. In his last 50 patients, the rate of recovery was 96 per cent. Castells *et al.*[14] treated a group of 21 patients, suffering from a variety of disorders such as tabes, sciatica, facial neuralgia, and tuberculous meningitis, with hydrocortisone intrathecally in 50 mg. doses every 48 to 72 hours, and attributed the favorable effects to the anti-inflammatory and antifibroblastic action, as well as the analgesic properties, of the steroids. The drug was found ineffective in a case of chronic spinal arachnoiditis.

MISCELLANEOUS SUBJECTS

Clinical investigation in neurosurgery, both in this country and abroad, apparently is concerned increasingly with neural physiology,[2] particularly as it is related to anesthesiology,[20,70] vascular surgery,[68,83] and the increasing use of hibernation in surgery.[37] Beduschi *et al.*[9] have reported their impressions of the role of the pyramidal tracts in the transmission of abnormal impulses, following a successful bilateral upper cervical tractotomy, after the technique of Putnam, for the treatment of a severe dystonia. Niswander *et al.*[62] in a series of 92 patients treated by lobotomy (method not described), claimed improvement in 3 out of 4 patients, but, as so many reports reveal, there was no control series studied. Hoerster and Jaco[40] studied 21 patients operated upon by transorbital leucotomy, and matched them on age, sex, diagnosis and length of hospitalization with a group receiving electric shock therapy and insulin coma therapy in another state hospital. Distinctly better results were obtained in the leucotomized patients, not only as judged by a variety of clinical factors, but also in eleven functions of the Malamud-Sands scale.

COMMENT

Scanning world-wide efforts in neurosurgery over the past several months leaves a few definite impressions. There is continuing interest in the pathology and surgical treatment of all varieties of intracranial vascular anomalies, aneurysms particularly, and much that has to do with new operative technique and instrumentation is in this general field. There continues to be a less lively interest in study of intracranial tumors in the United States, except that they constitute nuisances that must be met, and then often with ultimate defeat. However, there is still much active investigation of brain tumors reported in the foreign literature, particularly that coming from Germany,[26] France, and Scandinavia. Most local interest in brain tumors is found among the pathologists who publish their material in journals dealing with experimental and clinical neurology and pathology. There is a steadily growing interest everywhere in the surgical approaches to the basal ganglia, and though, at this early time, some of them seem rather wild and dangerous a dream, neurosurgeons, above all others, should have learned that the "impossible" may well become "possible," even commonplace. Most publications having to do with hypotension and hypothermia as surgical aids are from American sources. There is a remarkable falling off in publications having to do with leucotomy by any method.

It is regrettable that more Russian medical and surgical literature is not readily available in this country in English translation. Such translation is not only difficult to come by, but also when available is of prohibitive cost. Yet such publications as *Voprosy Neirokhirurgii* and related journals are rich in current Russian thought on

surgical technique, physiology of the central nervous system, the chemical and electrophysical properties of the brain, and new apparatus for surgery and physiologic study. State-subsidized work in neurosurgery and neurology in Russia is currently in an active and productive stage.

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CASE REPORTS

CONGENITAL ATRESIA OF THE FORAMINA OF LUSCHKA AND MAGENDIE WITH HYDROCEPHALUS

REPORT OF A CASE IN AN ADULT

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(Received for publication August 6, 1957)

Hydrocephalus as a result of congenital atresia of the foramina of Luschka and Magendie of the 4th ventricle was first described adequately by Dandy and Blackfan.[3] The subsequent reports and evaluation by Taggart and Walker[6] stimulated increasing interest in this disorder as reflected in the more recent literature. In only one fully authenticated case has the patient been an adult.[7] We wish to add a second.

CASE REPORT‡

A white man, 31 years of age, had been admitted to hospital on numerous occasions because of intermittent headache and blurred vision.

Family History. No pertinent history, although the patient's mother was said to have "heart trouble."

Past History. Bouts of pneumonia occurred when the patient was 18 months, 6 years and 9 years of age, respectively. At 9 years he also had diphtheria, and later measles, pertussis, influenza, and possibly mumps. There were no known complications of these diseases. Venereal disease was denied, as was also tuberculosis. At 13 he suffered a blow to the right parietal area in a sledding accident, and was said to have had a fractured skull. After a week of unconsciousness, he noticed tinnitus and marked loss of hearing in the right ear. The tinnitus soon cleared, but the loss of hearing persisted.

After enlisting in the Army in January 1943, the patient had frequent sore throats and occasional exertional dyspnea. He was admitted to hospital on July 3. His tonsils were found hypertrophied and cryptic. The tympanic membranes and the eyegrounds were normal, as was the remainder of the physical findings. The blood leukocytes were 14,200 per c. mm. Leukocyte differential, urinalysis and bleeding and clotting times were within normal limits. The Wassermann test was negative. Audiograms revealed a nerve type of deafness on the right with some loss for high tones on the left. Following tonsillectomy on July 8, there was an elevation of temperature which responded to sulfathiazole. The patient was dismissed from hospital on July 21.

He was admitted again on April 16, 1944, complaining of precordial pain and a slightly productive cough. He had had an upper respiratory infection 2 months previously, following which he had mild exertional dyspnea. His temperature was 101°F. and pulse was 108. Pleuropericardial friction rub and cardiac enlargement were noted. Leukocytes were 12,000 per c. mm. and erythrocyte sedimentation rate was 33 mm. per hour. Roentgenograms showed evidence of copious pericardial effusion. Rheumatic pericarditis being suspected, the patient was kept in bed and given salicylates.

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On being transferred to a convalescent center in Arizona on June 15, the patient's temperature and pulse were normal. No abnormalities were found on physical examination. Leukocyte count was 11,550 per c. mm. (76 per cent polymorphonuclear neutrophils, 5 per cent eosinophils, 5 per cent stab forms and 14 per cent lymphocytes), hemoglobin 85 per cent, erythrocytes 4.42 million, packed cell volume 36 per cent, and sedimentation rate 30 mm. per hour. The urine was normal except for occasional clumps of polymorphonuclear neutrophils and, on one occasion, several waxy casts. The electrocardiogram revealed T-wave changes which were interpreted as evidence of myocardial damage. Roentgenograms of the chest revealed cardiac enlargement and moderate pleural effusion. Nose and throat cultures were repeatedly negative for beta hemolytic streptococci. Bed rest and intensive salicylate therapy were continued for 2 months. By December 20, the electrocardiogram, erythrocyte sedimentation rate and pulse were all normal. At that time there was moderate cardiomegaly, as shown on roentgenokymograms and an esophagogram. The patient was discharged from the hospital and the Army on Jan. 18, 1945. At no time during his illness had he suffered from involvement of joints or had there been evidence of disease of the central nervous system.

Present Illness. In January 1952, when 31 years of age, the patient first noticed periodic blurring of vision in both eyes. The following month he had attacks of diffuse headache associated with weakness and numbness of the right leg. Slight nausea in the morning hours appeared in March.

He was admitted to hospital April 10 because of "sinusitis." Following submucous resection, the headaches and visual blurring became more severe. After leaving hospital on April 12, the patient continued to have attacks of headache and numbness and weakness of the right leg. New complaints appeared in the form of diplopia, positional vertigo, tinnitus in the left ear, and numbness over the left lower lip and chin.

The patient was readmitted on May 9, 1952, complaining of blurred vision bilaterally. Examination revealed regular but dilated pupils, bilateral papilledema of unspecified degree, and diminished but equal quadriceps-femoris reflexes. Leukocytes were 12,750 per c. mm., with 63 per cent polymorphonuclear neutrophils, 35 per cent lymphocytes and 2 per cent eosinophils. Lumbar puncture revealed clear, colorless, cell-free fluid with an initial pressure of 255 mm. of water. Protein content was not determined.

Further Examination. On May 13, at another hospital, the heart was questionably enlarged to percussion. Hearing in the right ear was found to be reduced about 50 per cent. There was pronounced papilledema bilaterally, and slight nystagmus on lateral gaze, particularly to the left. The abdominal reflex on the right side was absent.

1st Operation. On May 20, ventriculograms revealed symmetrical dilatation of the entire ventricular system and absence of air over the cerebral hemispheres. Under general endotracheal anesthesia, a suboccipital craniectomy was then performed. The cerebellar tonsils were found herniated into the foramen magnum. Elevation of the tonsils released copious spinal fluid. The inferior vermis was split, exposing a thin membrane over the roof of the ventricle. The membrane was opened widely, exposing the floor of the ventricle and the caudal end of the aqueduct, which was enlarged. Free communication between the basal cisterns and the 4th ventricle having been established, the incision was closed.

Postoperative Course. No complications developed. Repeated lumbar punctures revealed a gradual decrease of cerebrospinal fluid pressure from 310 mm. water postoperatively to 185 mm. water a month subsequently. Papilledema had receded at the time of the patient's discharge from the hospital on June 19.

Interval Course. After several days of well-being, nausea and vomiting set in, and there were bifrontal headaches, staggering gait, and further decrease in visual acuity.

Further Course. The patient was readmitted on July 1, 1952. He was alert and in no distress. Temperature was normal, blood pressure 118/80, and pulse 88. Papilledema was again evident. The left quadriceps-femoris reflex was decreased, and unsteadiness of gait with a tendency to veer to the left was noted. The Romberg test was negative. The region of suboccipital decompression was soft. The cerebrospinal fluid showed 250 mg. per cent of protein with

a cell count of 44 per c. mm., practically all lymphocytes. No organisms were recovered.

On July 2, ventriculograms were much the same as previously, except that air was present in the cisterna magna. The right lateral ventricle was again punctured. Approximately 80 cc. of air were removed and 1.5 cc. of indigo-carmine dye mixed with warm saline were injected. A Tuohy needle was then inserted into the lumbar sac. Fluid flowed freely, but after 40 minutes no dye had appeared. The needle was replaced by a catheter, which was connected to a syringe with a sterile rubber glove for a reservoir. Seventy minutes after its injection into the ventricle, the blue dye appeared in the spinal fluid draining from the catheter.

Fluid drained from the catheter for 10 days in amounts averaging 60 cc. per day. The fluid was at first dye-stained, then xanthochromic, and finally clear and colorless. The headaches disappeared, but the patient did not notice any substantial improvement in visual acuity. On July 12 the catheter was removed.

Pneumoencephalogram on July 15 revealed air in many of the subarachnoid pathways and a small amount in the right lateral ventricle. Subsequently, the patient remained afebrile, headaches were much less severe, and papilledema had decreased. He was discharged from hospital on July 23.

By December 15, the patient's vision had improved to an acuity correctible to 20/25 bilaterally. Funduscopic examination showed that "the left optic disc is almost flat, the right disc still evidences residual edema." Soon thereafter the patient's vision began to fail, papilledema increased, and vomiting set in.

Further Admission, Jan. 10, 1953. The most outstanding abnormalities were unsteady gait with faulty equilibrium, bilateral old papilledema which was most marked on the right, and nystagmus on lateral gaze, especially to the right. Pulse was 88 and regular; blood pressure 135/80. Cerebrospinal fluid: February 9, total protein 29 mg. per cent, no leukocytes; May 4, total protein 90 mg. per cent, 9 leukocytes per c. mm. The visual fields were moderately constricted.

2nd Operation. On February 9, reexploration of the posterior fossa was performed under general endotracheal anesthesia. The posterior inferior part of the cerebellar vermis was again divided. The 4th ventricle was dilated to much the same degree as at craniotomy in 1952. A thin membrane obstructed the flow of fluid from the ventricle. The cerebellar tonsils had herniated into the foramen magnum and were bound down by adhesions. The cisterna magna and the 4th ventricle were opened widely and the cerebellar tonsils were amputated. Free communication of the ventricular and subarachnoid spaces having been accomplished, the incision was closed.

Course. Several lumbar punctures were performed, with the fluid pressure gradually falling to normal. The patient felt well enough to be released from hospital on March 6. When seen on April 6, he reported that his vision had improved and that he had been free from headache.

Final Admission. On May 9, 1953, the patient returned to hospital complaining of nausea, pronounced blurring of vision, and severe occipital headaches. Improvement had been sufficient for him to resume his job as a truck driver, but after several days of work his symptoms recurred. He walked with a wide-based, staggering gait, and the Romberg test was positive.

The patient rapidly became comatose. On May 11 the right lateral ventricle was punctured and 100 cc. of clear cerebrospinal fluid under high pressure were allowed to escape. The patient became lucid. Indigo-carmine dye was then injected into the ventricle and lumbar puncture was performed. After 25 minutes, only a trace of the dye had appeared in the spinal fluid. Several hours later the patient lost consciousness and death quickly ensued. The illness, from the time of development of symptoms of increased intracranial pressure, had lasted 16 months.

Autopsy. Dense fibrous pleural adhesions were noted over the upper lobes of the lungs, and there were fibrinous adhesions at the base of the lungs and between the heart and pericardium. The lungs were moderately edematous. The heart weighed about 500 gm. and was slightly dilated. The wall of the left ventricle was about 1.75 cm. and that of the right ventricle was 0.5 cm. at the points of their greatest diameter. The cardiac valves were normally

formed and without gross lesions. Although there was an acute inflammatory exudate about one of the larger coronary arteries, no evidence of rheumatic fever was found on either gross or microscopic examination of the heart.

Brain, Gross. The cerebrum appeared somewhat swollen; its surface was dry. The entire ventricular system was uniformly dilated. On removal of the brain the cerebellum appeared swollen. Abundant fibrous tissue was present over the roof of the 4th ventricle (Fig. 1). The foramina of Magendie could not be identified.

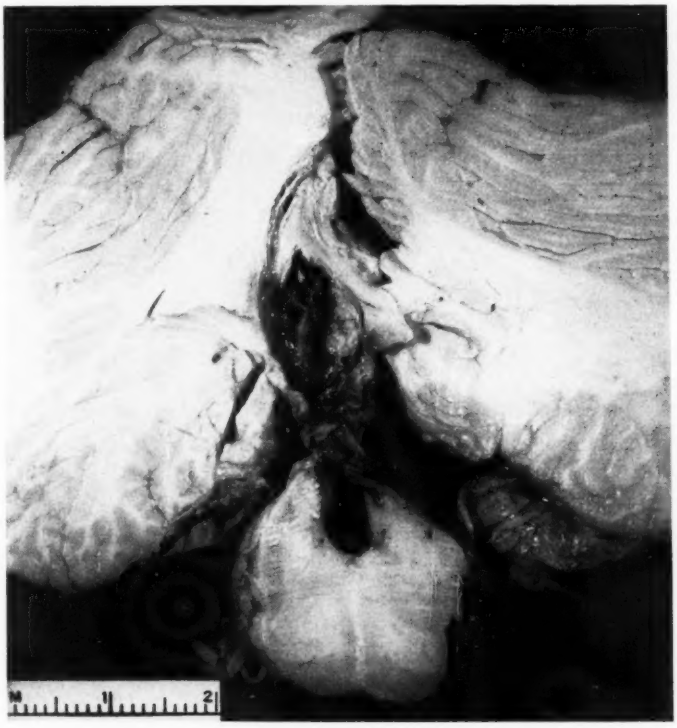

FIG. 1. Medulla oblongata and cerebellum, showing pigmented cyst over the roof of the 4th ventricle and part of the cyst wall attached to the cerebellum on each side. (AFIP neg. #54 8197)

Emanating from the cerebellopontile angle and attached firmly to the medulla oblongata and cerebellum on each side was a fluid-filled cystic structure measuring approximately 2 cm. in diameter. Superiorly, each extended to the level of the facial nerves, to which they were adherent, while inferiorly each reached the uppermost spinal cord. On sectioning, both cystic structures were found continuous with the foramina of Luschka, and, through them, with the much dilated 4th ventricle (Fig. 2). Within them was clear dye-stained liquid which obviously was cerebrospinal fluid. Tufts of choroid plexus were found in the roof of the ventricle and along the inner wall of both of the cystic structures. Small hemorrhagic areas were present in the wall of the ventricle, especially on the right side. The block of tissue containing the lateral recesses of both sides was lost subsequently.

Microscopic Examination. Sections from several areas of the cerebral hemispheres showed slight thickening of the leptomeninges and occasional hemosiderin-filled macrophages. Scanty

hemorrhage was noted at the site of ventricular puncture, and a small hematoma as well as parenchymal softening in the region of the needle track. The choroid plexuses of the lateral ventricles were unaltered.

Section through the medulla oblongata near the level of the lateral recesses revealed moderate subependymal edema and several small hemorrhages in the ventricular wall. Ependymal epithelium on the part of the lateral recesses available was continuous peripher-

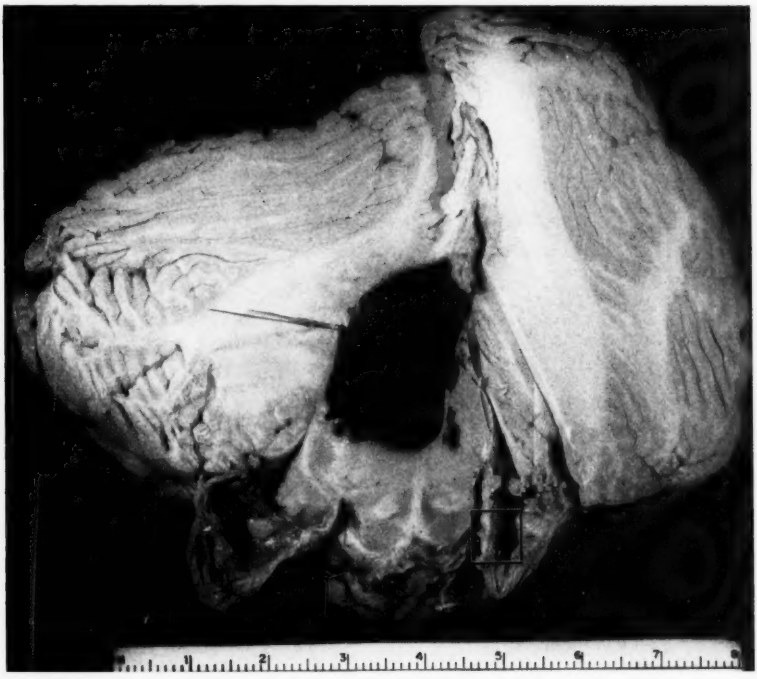

FIG. 2. Brain stem and cerebellum, showing hemorrhagic areas in the wall of dilated 4th ventricle and cysts continuous with the lateral recesses. Tufts of choroid plexus are present within the cysts. (AFIP neg. #54 8199)

ally and completely lined the cyst walls (Fig. 3). Much of the wall proper was composed of neuroglial tissue having the same structure as that of the wall of the lateral recesses. Externally, the cyst walls were fused with thickened arachnoidal tissue. The foramina of Magendie could not be identified. Because of the dense fibrous tissue resulting from the two operations performed in that area, what remained of the roof of the 4th ventricle was composed almost entirely of fibrous tissue.

DISCUSSION

The ependymal and glial nature of the cyst walls and the direct continuity of the cysts with the lateral recesses and 4th ventricle, as noted grossly, seem to constitute sufficient evidence that the cysts represent congenital anomalies. These features rule out the possibility that the cysts arose as a consequence of basal cisternal arachnoiditis secondary to the cranial trauma or the rheumatic disease from

which our patient allegedly suffered. Basal cisternal arachnoiditis, as discussed by Horrax,[5] is easily differentiated from the lesion in our case.

The assumption is that atresia of the outlets of the 4th ventricle existed congenitally and that as a consequence of intraventricular pressure the atretic ends of the lateral recesses gradually ballooned. Porosity of the cyst wall must have been such that ample fluid filtered through it during most of the patient's life. The eventual internal hydrocephalus may have been caused by increasing leptomeningeal fibrosis about the cysts, as well as over the foramina of Magendie.

We could find no evidence that the vermis was maldeveloped. As a rule, congenital atresia of the outlets of the 4th ventricle with ensuing hydrocephalus (with or without cyst formation as in our case) is associated with dysplasia of the inferior

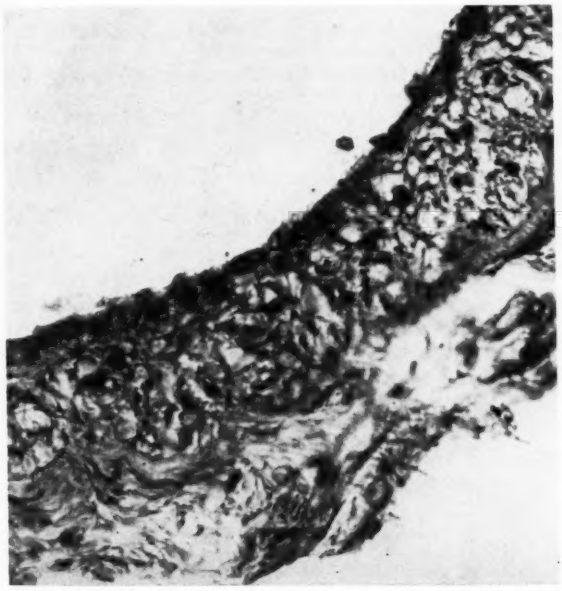

FIG. 3. Section of cyst wall indicated in Fig. 2, showing the ependymal nature of lining of the cyst. Phosphotungstic acid hematoxylin stain, ×440. (AFIP neg. #56 19677)

vermis of the cerebellum, a form of rachischisis. As brought out by Taggart and Walker,[6] this disorder usually occurs in early youth and is characterized roentgenographically by enlargement of the posterior fossa, elevation of the transverse dural sinuses, and cystic dilatation of the 4th ventricle. There may also be papilledema. Cases of this kind have also been described by Benda[1] and more recently by Gibson.[4] Occasionally the dural sinuses are not displaced[2] and the vermis of the cerebellum is normally formed, as demonstrated in our case.

SUMMARY AND CONCLUSIONS

This report concerns a case of hydrocephalus in an adult, associated with large cystic structures in the cerebellopontile angles, the walls of which were continuous

with the 4th ventricle through the lateral recesses. From the histological structure of the cyst wall, as well as for other reasons, the cysts were regarded as outpouchings of congenitally atretic foramina of Luschka. It is assumed that the foramina of Magendie were also congenitally atretic.

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NOTE

Reprints may be obtained from Armed Forces Institute of Pathology, Washington 25, D. C.

NOTICES

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

The winner of the Fourth Annual American Academy of Neurological Surgery Award is Dr. Norman Hill, Mayo Clinic, Rochester, Minnesota, for a paper entitled "Cerebrospinal Fluid Proteins, Glycoproteins and Liproproteins in Obstructive Lesions of the Central Nervous System."

The Academy announces its Fifth Annual Award. The award is intended to stimulate research by younger neurosurgeons during or immediately following their training. It will be given for the best paper submitted reporting original clinical or laboratory research pertaining to the nervous system, for which the author has had primary responsibility. The paper shall not have been accepted for publication in any journal, but may have been submitted as a thesis for degree. Eligibility is confined to doctors of medicine currently in training in neurological surgery, or those who have completed their training since June 30, 1957. Those certified by the American Board of Neurological Surgery, The Royal College of Surgeons of Canada, subspecialty neurosurgery, or with other equivalent certification are not eligible.

The recipient of the Award will be invited to present his paper at the 21st Annual Meeting of the Academy at Pebble Beach, California, October 18–21, 1959. Expenses to and from the meeting (from port of entry to the United States if living outside the North American Continent), expenses while attending the meeting, and an honorarium of $100.00 will be paid the recipient.

Manuscripts should be sent to Dr. Robert McLaurin, Cincinnati General Hospital, Cincinnati 29, Ohio, before July 15, 1959. A resume of the candidate's postdoctoral training must accompany the paper when submitted.

MEETINGS

American Academy of Neurological Surgery, Royal York Hotel, Toronto, Canada, Nov. 6–8, 1958. Dr. Robert McLaurin, Cincinnati General Hospital, Cincinnati 29, Ohio, Secretary.

American Neurological Association, Claridge Hotel, Atlantic City, N. J., June 15–17, 1959. Dr. Charles Rupp, 133 South 36th St., Philadelphia, Pa., Secretary.

Association for Research in Nervous and Mental Disease, Hotel Roosevelt, New York City, Dec. 12–13, 1958. Dr. Rollo J. Masselink, 700 West 168th St., New York, 32, N. Y., Secretary. (Subject: Neuromuscular disorders.)

Harvey Cusing Society, Roosevelt Hotel, New Orleans, La., April 30–May 2, 1959. Dr. David L. Reeves, 316 West Junipero St., Santa Barbara, Calif., Secretary.

International Congress of Neurological Surgery (II), Statler Hotel, Washington, D. C., Oct. 14–20, 1961. Dr. Bronson S. Ray, 525 East 68th St., New York 21, N. Y., Secretary-General.

Latin American Congress of Neurosurgery, Santiago, Chile, April 12–15, 1959. Dr. Alfonso Asenjo, Casilla 70 D, Santiago, Chile, Secretary-General.

Neurosurgical Society of America, The Homestead, Hot Springs, Va., March 4–7, 1959. Dr. Frank P. Smith, 260 Crittenden Blvd., Rochester 20, N. Y., Secretary.

Southern Neurosurgical Society, Americana, Bal Harbor, Miami Beach, Fla., Jan. 23–24, 1959. Dr. C. D. Hawkes, 22 N. Manassas St., Memphis 5, Tenn., Secretary.

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SUBSCRIPTIONS. The subscription rate for the volume of six bi-monthly issues is $11.00 in the United States and Pan-American Union; $11.50 in Canada; $12.00 in other countries. Single copies are $2.00 postpaid. Single copies of Volume VIII, Number 1, January 1951 issue: A Symposium on Intracranial Vascular Abnormalities: $3.00 postpaid, if available. Volume I, Number 1 is supplied only on orders for the complete volume. Payments may be made in advance to Charles C Thomas Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois, U.S.A.

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George Banta Company, Inc., Menasha, Wisconsin, U.S.A.